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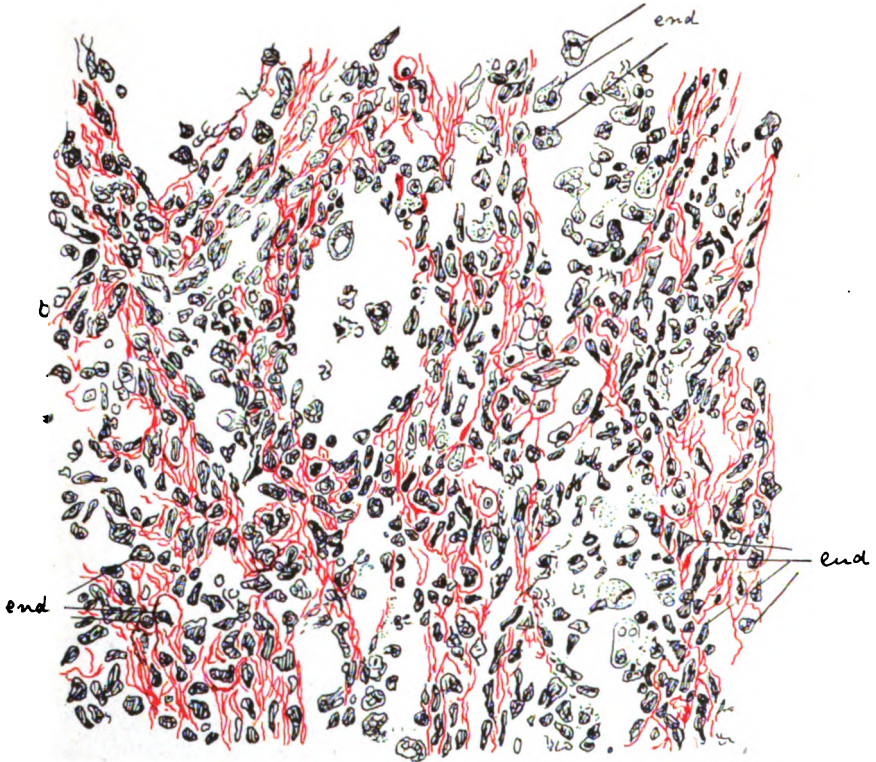


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FIG. 3.



Histological picture of tumor from aortic valve.

Pages 155, 156.

INTERNATIONAL CLINICS

A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-
RICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS,
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,
OTOLOGY, RHINOLOGY, LARYNGOLOGY,
HYGIENE, AND OTHER TOPICS OF INTEREST
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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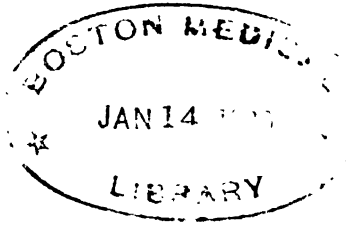
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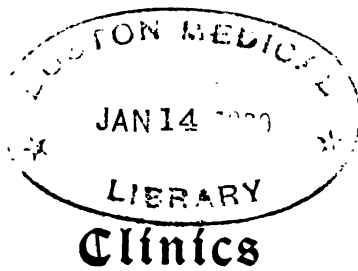
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MALARIA, WITH SPECIAL REFERENCE TO ITS TREATMENT AND CONTROL

DELIVERED AT THE COLLEGE OF MEDICINE, UNIVERSITY OF ILLINOIS, JULY 16, 1919

By C. C. BASS, M. D.

Professor of Experimental Medicine at Tulane University, New Orleans,
Louisiana

DR. DAVIS J. DAVIS, Dean of the Medical Department, in introducing Doctor Bass, said: It has been said that the fall of Rome was due to malaria. I suppose we are apt in northern climates to forget the great importance of malaria as a problem, not only medically, but economically and industrially.

Doctor Bass has been investigating this disease and has been making a special study of it for many years, devoting practically all of his time to studying the methods of control on a large scale of this scourge, and he is here to-day to tell us about his investigations and his attempts along these lines, and we are very glad indeed to welcome Doctor Bass to the University of Illinois. I take great pleasure in introducing to you Dr. C. C. Bass, Professor of Experimental Medicine in Tulane University, New Orleans.

DOCTOR BASS said: *Mr. Dean, Ladies and Gentlemen:* I take great pleasure in talking to you this afternoon on the subject of malaria, to which I have devoted considerable of my time and study for the last few years.

Malaria is caused by an animal parasite which must have man as one of its hosts. The only other host of malaria parasites is certain kinds of mosquitoes. Both of these hosts are necessary, and the parasite species cannot be perpetuated without both of them. Malaria is generally thought of as being a disease in which there are certain definite symptoms. This idea is somewhat misleading. Please let us for the present look upon it as an infection with parasites by which recognizable symptoms may or may not be produced.

When malaria parasites from the same source are introduced into different individuals, they may and do produce quite different effects. For instance the parasites may multiply and give rise to certain clinical symptoms, of which periodical chills and fever are the most prominent. These symptoms may be continued for days or weeks, never, however, becoming alarming. Such a case, perhaps, would be spoken of as ordinary chills and fever or an ordinary attack of malaria. Other individuals, infected perhaps from the same source and at the same time, may develop much more severe symptoms, having the so-called congestive chill, comatose or other pernicious form of malaria, and even death may result. Usually in such instances the number of parasites present is very much greater than in the less severe form. Still another individual, infected perhaps from the same source and at the same time, may not have any recognized symptoms of disease. He feels fairly well, and no examination that we can make may demonstrate any definite deviation from normal health.

If we examine his blood we may find a few malaria parasites from time to time, and at other times we may not be able to find any parasites. He may go on for many months, and finally lose his infection entirely, without ever having any symptoms of disease that he or anybody else recognizes or can demonstrate. There is no question but that a certain amount of damage must be done by parasites even in such small numbers, though we cannot demonstrate the damage by methods in use at the present time. Such an infected individual has enough resistance against the parasites to prevent multiplication in sufficient numbers to produce clinical symptoms. If, however, something occurs which lowers his general vitality, such, for instance, as sudden chilling of the surface of the body, violent exertion, overeating of indigestible food, the shock of surgical operations, etc., he is then likely to begin to have clinical symptoms of malaria. His resistance has been lowered sufficiently to permit the parasites to multiply more rapidly.

In order to get the manner of growth and the life cycle of the parasites well before us, we will make use of the lantern. Here (pointing to pictures) are young malaria parasites in blood-cells. You will note that each parasite consists of a mass of cytoplasm and a smaller nucleus or chromatin granule. The parasites are all attached to, or are within, blood-cells, and remain so throughout their life cycle.

These are the youngest malaria parasites and it will be well for us to follow their development to full maturity and reproduction in order to get a clear conception of the manner in which they reproduce and especially the manner in which the symptoms of the disease are produced.

In the next slide we have represented parasites that are about twelve hours old. You will note that they have grown in size, both the cytoplasm and nucleus, and that they are still more or less ring-shaped as they were at first. Up to this stage they are frequently spoken of as signet rings because of their shape. We do not notice any remarkable change except in size. All parasites have grown considerably and are now almost twice as large as they were at first.

In the next slide we have represented parasites about twenty-four hours old. You will note that they have more or less lost the signet-ring appearance and have grown considerably more in size. Many of the parasites are now more than one-half the diameter of the red blood-cells in which they are growing. Not only have they grown in size, but their nuclei have become polymorphous. Many of them have elongated and some are much narrower at the middle than they are at each end. In addition to the change in size and the polymorphous appearance of the nucleus, we observe a few small brownish pigment granules scattered in one or another portion of the parasite. It is difficult to say whether the granules are in the parasite or on it, but they are closely associated with it. These pigment granules have been produced by the breaking down of the hæmoglobin of the blood-cell by and appropriation of it, or certain portions of it, for the nutrition of the parasite. It is uncertain whether hæmoglobin is the only nutriment of the parasite or whether it simply serves as a part of it. It is broken up into globin and hæmatin, the globin going to produce the body of the parasite and the hæmatin forming these insoluble pigment granules. As the parasite grows still further, more pigment is produced.

In this slide we have represented parasites about thirty-six hours old. You will note that they have grown still larger and have now almost consumed the blood-cells in which they grew. In some instances there remains little more than a thin capsule of blood-cell substance surrounding the parasite. Not only has the cytoplasm of the parasite continued to grow, but the nucleus has become more

polymorphous and has divided into a number of portions. In some we can make out as much as four, six, eight or ten divisions. Some of these subdivisions, we can see, are preparing to divide still further. The pigment which a few hours previously was distributed throughout every portion of the parasite has now collected in a mass. These masses of pigment are usually located more or less eccentrically in the parasite.

In this next slide we have represented parasites approximately forty-eight hours old. Most of them have reached full maturity. You will note that each parasite has continued to grow until now it is almost as large as a red blood-cell and that it has consumed the cell in which it grew until nothing but a thin capsule remains. If we examine carefully we find that the nucleus of the parasite has continued to divide until now there are from twenty-four to thirty-two divisions. The cytoplasm has also divided so that each nuclear division has its own cytoplasm. In fact, we have twenty-four to thirty-two young malaria parasites within the capsule in which the parent parasite developed. Each one of these young parasites consists of a nucleus and its own cytoplasm and each one under favorable conditions is capable of attaching itself to another red blood-cell and growing through a cycle of development similar to that through which the parent grew. Just at this time the capsule in which the parasite developed has become so thin that it ruptures at one place or another and sets free these young parasites, known as merozoites. Though each young parasite is capable of attaching itself to another blood-cell and growing to maturity and reproducing in the same way, as a matter of fact, most of them are soon destroyed after they are set free.

Two agencies, at least, destroy them. One is the phagocytes and the other a lytic substance in blood plasma and serum. Only a small portion succeed in attaching themselves to red blood-cells and thereby protecting themselves from these destructive agencies. In cases of pernicious malaria a greater proportion of parasites grow. In cases in which recovery is taking place and the infection is being whipped out, even less than one parasite for each adult lives.

There are three different species of malaria parasites known and differentiated, the *tertian*, *quartan* and *estivo-autumnal*. We have only two, however, in this country, except in rare instances in which cases of quartan are imported and also in a few limited areas in which

quartan malaria does prevail. My impression is that there is more quartan malaria in certain sections of Arkansas and Tennessee than in any other States in the United States. For all practical purposes we may make no differentiation of the species in our discussion of the manner in which they produce disease.

There is a tendency for each species of malaria parasites to reproduce by crops. The tendency is for all parasites that will segment on a given day to segment at about the same time of day. This tendency is more pronounced in tertian and quartan malaria than in estivo-autumnal. In consequence of this tendency in cases of malaria infection in which there are sufficient parasites to give rise to clinical symptoms, countless millions of parasites segment at about the same time of day, setting free, of course, these countless millions of merozoites, or young parasites.

Not only do they set free these countless millions of young parasites, but the remaining contents of the blood-cell capsules in which the parasites grew are set free. This consists, at least, of large amounts of pigment and certain other substances and débris. It is highly probable that the substance (or substances) with which the parasite has been able to break down the blood-cell and appropriate it for its nutrition is an important factor in the production of clinical symptoms. Something produced at the time of segmentation of the parasites, whenever in sufficiently large amount, is toxic and produces definite effect upon the system.

One of the first effects of this toxic substance, whatever it is, when produced in sufficient quantities, is to cause a general relaxation of the entire vascular system. As a result, the blood of the body tends to accumulate more or less in the large vessels and organs of the body, and little blood goes through the peripheral circulation. As a result, the skin is not kept warm in the natural way and it takes on the temperature of the surrounding air and gets cold, and the patient has a chill.

The chill lasts a variable length of time, depending, no doubt, upon several different factors, among them being the amount of toxin produced, and the ability of the individual to react from its effect. In ordinary intermittent malaria the chill usually lasts from a few minutes to half an hour or a little longer.

Another effect of this toxic substance is to cause elevation of tem-

perature, or fever. This is a more certain effect, perhaps, than that of chilling of the body. It sometimes occurs that patients have the fever without the chill, but it never occurs that malarial chills occur without fever. If the temperature is taken per rectum, it is found that the fever comes on during or even before the occurrence of the chill, but of course the surface of the body does not get hot until after the chill has passed off. The fever lasts a variable length of time, depending upon the ability of the individual to neutralize or eliminate the toxin, but usually only a few hours at most. The temperature returns to normal nearly always with a profuse sweat. It remains at about normal until forty-eight hours from the time the first chill occurred, when another crop of parasites segment and another chill and fever occur. Every other day malarial chills and fever are caused by the segmentation of a crop of malaria parasites at or about the time the paroxysm sets in. The patient feels fairly well between the paroxysms except, of course, more or less "used up" from the experience.

Sometimes an individual may be infected with two crops of parasites, one segmenting every other day and the other on the alternate days, giving rise then to a chill and fever every day. In such an instance the patient has two cases of malaria at the same time.

The life cycle of quartan malaria parasites is seventy-two hours instead of forty-eight, and therefore paroxysms occur every seventy-two hours. Estivo-autumnal parasites do not reproduce by crops as definitely and certainly as do the tertian and quartan parasites and often there is more or less segmentation taking place continually, resulting in continued damage and therefore continued production of toxins and continued fever. Practically, all continued malarial fever is produced by estivo-autumnal infection. It is to be remembered, however, that estivo-autumnal infection may also cause intermittent fever. It is interesting to note that estivo-autumnal infection causes practically all the pernicious malaria and deaths that occur from malaria. Tertian and quartan malaria seldom produce death.

You may be interested in an explanation of the fact that estivo-autumnal parasites are so much more destructive than the others. Tertian and quartan parasites are more or less ameboid and therefore can and do pass through the smallest capillaries without great difficulty. They do not lodge in these capillaries to any great extent.

On the other hand, estivo-autumnal parasites have little ameboid activity and they seem to be considerably firmer in consistency. The parasite is round or oval and rather unyielding to pressure. By the time an estivo-autumnal parasite is about twelve hours old it lodges in a capillary blood-vessel somewhere in the body. On account of this firm or unyielding consistency, it remains in the capillary during the balance of its life. Those of you who have examined the blood of persons infected with estivo-autumnal parasites will recall that the only form of parasite you see is the small ring form. These are seldom more than twelve hours old. It is true you may see some gametes or crescents, but these are of such shape and consistency that they do not lodge in the capillaries like the asexual parasites do.

These parasites lodge in narrow places in capillaries, the lumen of which, as you know, is more or less irregular in shape. Whenever the parasite reaches full maturity and segments, of course the young parasites are swept out into the general circulation again.

Malaria parasites tend to lodge in certain organs and tissues of the body more than they do in others, the spleen, perhaps, receiving the largest number. In certain individuals and for reasons which we do not at present fully understand, they sometimes lodge in very large numbers in such organs as the brain, kidney, stomach, intestines, etc. Whenever this occurs, it gives rise to the particular clinical form of malaria in which symptoms affecting these particular organs predominate. Sometimes sufficient parasites lodge in the capillaries of the brain to greatly interfere with the circulation of blood through them. As a result of the anæmia of the brain, the patient becomes comatose and we have what is known as comatose malaria. It is interesting to note that perhaps 50 per cent. of all deaths from malaria occur in comatose cases. It is therefore a very serious clinical form.

After an individual has been infected for a period of at least two or three weeks or longer, usually if we examine the blood carefully we may find a few, or sometimes many, parasites which are different from those which have just been described and which are recognized by those who are familiar with them as being sexually differentiated parasites. They are male and female, whereas those we have previously described are schizonts, and they are not capable of reproducing in the body of man. They probably do not produce any symptoms and the length of their life is about two or three weeks. If blood

containing them is drawn, however, by favorable species of mosquitoes, they may then reproduce by sexual activity, giving rise to sporozoites, a very small form of parasite, which collects in the salivary glands of mosquitoes. They may live in the glands of the mosquitoes for several days, or perhaps weeks, and will be inoculated in any person from whom the mosquito draws blood.

As long as a person has these sexually differentiated parasites (gametes) in his blood, he is likely to be a source of infection to others. There are times when persons who have malaria have no gametes in their blood or at least not sufficient to infect mosquitoes. At other times the same individual may have many gametes and may be a source of infection. Therefore, any person who has malaria is a potential source of spread of the disease as long as he remains infected, and the only certain way of rendering an individual no longer a potential source of infection is to disinfect him of his parasites. Fortunately, we have a perfect specific with which this end can be accomplished with absolute certainty.

It is interesting to note the large number of people living in a region where malaria is very prevalent who are infected and are therefore potential sources of infection to others without having any symptoms whatever. In a recent investigation of malaria in Bolivar County, Mississippi, by the International Health Board under the auspices of the Mississippi State Board of Health, it was found that a very large proportion of those who actually had malaria parasites in their blood presented no symptoms of the disease at the time and in many instances had not had symptoms previously that had been recognized as malaria. Analysis of the data on 31,459 persons whose blood was carefully examined and whose history as to previous attacks of malaria was recorded has brought to light some valuable information. Those who had had attacks of malaria or symptoms of any kind which were believed to be due to malaria during the previous twelve months were recorded as positive histories. Those who had had no symptoms at all were recorded as negative. The blood was taken at the same time and examined by the thick film method, by which the largest per cent. of the actual infection is found that can be found by any practical method of examination. Of the total number found to have parasites in their blood, only a little over 55 per cent. gave positive histories of attacks of malaria during the previous twelve

months and a little over 44 per cent. gave negative histories. This indicates that at least 44 per cent. of the malaria in that region, therefore, produces no symptoms whatever or does not produce symptoms that are recognized as being caused by malaria.

It frequently occurs that if one or more members of a family are sick with malaria, if we examine all persons living in the same home we will find parasites in the blood of others who are not sick at the present time and who have not been sick so far as they know. In fact, some of such infected individuals may remain infected for months and finally lose their infection without ever having any recognized symptoms produced by it. On the other hand, in the event anything should occur which greatly lowers the vitality or resistance of such an infected individual, he may then begin to have active clinical symptoms. The same thing occurs in individuals who have had attacks which have been relieved by treatment but who remain infected. They relapse from time to time as the result of recognized and sometimes unrecognized influences.

Overeating and eating indigestible food are among the prominent causes of occurrences of relapse in persons who have previously had attacks or in persons who have otherwise been carrying their infection without clinical symptoms. There is a common belief among people living in malarious sections that eating unripe watermelons, muscadines, etc., will cause chills and fever. As a matter of fact, they do not cause the infection, but they do precipitate attacks in persons who already have malaria.

This fact that persons may carry malarial infection for weeks or months without showing recognized clinical symptoms is of great importance both from the standpoint of the spread of the disease and also from the standpoint of treatment. Such infected individuals may at any time develop gametes in their blood and therefore spread the disease, even though they are not known to have it. If persons carry the infection without showing any recognized symptoms, it is very apparent that disappearance of symptoms can not serve as a guide as to when the patient has been disinfected. It is a mistake to suppose that as soon as a person who has had an attack of malaria is relieved of his clinical symptoms he is cured. Laymen think that, and many physicians do not realize the importance of continuing treatment until the patient is thoroughly disinfected. As a matter

of fact, only a small proportion of malaria cases treated by doctors are disinfected. Either the advice given the patient is not what is necessary, or it is given in such a way as not to impress the patient with the importance of carrying it out.

Not only is the disappearance of all symptoms not a dependable guide as to when the patient has been disinfected, but, unfortunately, even blood examination is not dependable. When there are no clinical symptoms present it frequently occurs that infected persons have not sufficient parasites in their blood at the time for them to be found by the usual methods of examination. They may be found at other times. The very fact, however, that they can not be found at all times reduces the value of a negative finding. Since we can not depend upon the disappearance of symptoms nor upon blood examination to determine whether an individual has been disinfected or not, we must depend upon some standard form and duration of treatment which has been shown by experience to disinfect a large per cent. of, if not all, malaria cases, and this is the chief fact which I wish to impress upon you this afternoon.

All effective treatment for malaria depends for its success upon quinin in some form or other. There is no other specific remedy for malaria known. The question that concerns us chiefly is the proper dose and proper duration of treatment. Extensive experiments conducted over a period of three years, in which many thousand cases of malaria were treated, employing different doses of quinin, and in a good many experiments different salts of quinin, have led me to conclude that there is no salt of quinin more effective in treating malaria than ordinary sulphate and that there is no method of administration more effective than administration of 10 grains for adults, daily, or a proportionate dose for children.

The length of time treatment is to be continued will depend upon what degree of effectiveness we wish to obtain. If, for instance, it is taken for only one week, very few cases are disinfected. If it is taken for a period of four weeks, perhaps 60 to 70 per cent. of cases are disinfected. If taken for a period of eight weeks, more than 90 per cent. of the cases are disinfected. To disinfect 100 per cent. would probably take between twelve and sixteen weeks. We have adopted eight weeks as the standard period, believing that this furnishes sufficiently satisfactory results for all ordinary purposes. We believe

that all cases of malaria should be given this amount of treatment for the purpose of disinfecting them after they have been relieved of the acute symptoms, by giving 30 to 40 grains, daily, for a period of three or four days.

If this eight weeks' treatment is carried out in all cases, it is true that a certain and considerable proportion of cases will be disinfected before the end of the period, and will therefore take more or less quinin in excess of what was actually needed. Since we have no method of determining when an individual has been disinfected, this can not be avoided. There is no serious objection to it, however, because of the fact that the small doses of quinin employed are not harmful and usually do not produce any considerable discomfort.

The question naturally arises in your minds—What will be done with the small per cent. who will relapse following this eight weeks' period of treatment? Understand, they do not relapse because quinin will not disinfect them of malaria, but simply because it does not do so when given in this size dose and for this particular period of time. Careful inquiry into the previous attacks will usually indicate those who are likely to be resistant and difficult cases to disinfect. Most of them will give a history of having taken considerable treatment previously and relapsing after it had been discontinued. There are very few first-attack cases that are not disinfected by the eight weeks' treatment.

If one thinks from the history or for other reasons that a particular case is likely to be difficult to disinfect, he should then advise somewhat longer than the eight weeks' period of treatment. Little harm will be done by adding a few more weeks for good measure. Patients are usually quite willing to accept and carry out the advice if they are properly informed of the reason therefor. Whenever a patient relapses after having taken treatment, no matter whether it was eight weeks or even longer, no credit on the length of treatment necessary to disinfect him can be given for whatever treatment was previously taken. Usually, when this fact is emphasized to patients who are likely to have relapses, they are quite willing to take quinin a little longer to make assurance doubly sure.

As evidence of the effectiveness of this method of treatment, I wish to call your attention to this map (pointing to picture). This is a map of an area of nine square miles in Bolivar County, Missis-

sippi, in which a malarial survey of all the people living in it was made. The red dots represent people who had malaria and the clear rings people in whom malaria was not found. A little more than 80 per cent. of all the people living in the area were found to have malaria. They were put upon the standard treatment just described to you and one year afterward another survey was made. There has been a reduction of a little more than 80 per cent. in the prevalence of malaria, and this next map shows how little malaria there was in the community compared with what was found before treatment. You will note how strikingly this map contrasts with the other one and it may serve to impress to some extent, at least, the effectiveness of the treatment employed in control of the disease.

A question of particular importance that arises in connection with the treatment of malaria with quinin is the form in which it shall be given and the time of day. It matters little whether the quinin is in solution, in capsules or tablets or otherwise prepared, so far as the effect upon the malaria is concerned. Quinin in acid solution, however, is so unpleasant to take that few people have the grit to continue it long enough to disinfect them of malaria. Any doctor who gives or contemplates giving quinin in this form should take a course of the treatment himself. He would then know better than to prescribe it for his patients.

The most convenient form for those who can swallow tablets or capsules is ordinary compressed tablets. They are perfectly satisfactory. For young children who can not swallow tablets or capsules well, we prefer quinin suspended in aromatic syrup of yerba santa. It should be so prepared that one teaspoonful contains the desired amount of quinin sulphate. It is rendered very much less unpleasant to take and children usually take it quite well. We advise that it should be given to children under eight years of age in this form.

The standard doses required to disinfect children of different ages, as well as 10-grain doses for adults, are as follows: Under one year, $\frac{1}{2}$ grain; one year, 1 grain; two years, 2 grains; three and four years, 3 grains; five, six and seven years, 4 grains; eight, nine and ten years, 6 grains; eleven, twelve, thirteen and fourteen years, 8 grains; fifteen years or older, 10 grains. These proportions are based upon calculations on results obtained in the treatment of several thousand people and should be followed in preference to doses

indicated by Young's rule or other rules for calculating doses for children.

The time of day at which the dose of quinin should be taken is immaterial so far as the effect upon the malaria is concerned. It is, however, very much more convenient for the patient to take it at night before retiring than at other times of day. This is especially true because of the fact that a large part of the treatment is carried out while the patient is up and about at his usual occupation. It also has the advantage that usually whatever unpleasant symptoms are produced have worn off before the next day and are not felt to the same extent during the active period of the day as they would be if quinin was taken at any other time.

I wish to make a plea against special and spectacular methods of treating malaria and for the adoption of this method which gives satisfactory and uniform results. Administration of quinin intramuscularly, hypodermically, and intravenously are seldom ever called for and they should not be employed to the neglect of the more effective and less spectacular administration by mouth. There are occasional pernicious cases of malaria in which it might be impracticable to give quinin by mouth at once, or in which the life of the patient might depend upon getting quinin into the circulation more quickly than it would be absorbed from the gastro-intestinal canal. In that case it should by all means be given intravenously. It reaches the blood-stream at once and does whatever good quinin can do. The cases in which it is required are extremely rare. Intramuscular administration is, in my judgment, seldom if ever indicated. If the patient needs quinin badly enough and there is any reason why administration by mouth can not be depended upon, then it should be given intravenously.

Neither intramuscular administration nor intravenous administration can be depended upon at all for the purpose of disinfecting the patient. It is practically impossible to disinfect a patient by either method, because it is not practical to carry out treatment by either method long enough to disinfect, whereas it is always practical to disinfect by ordinary administration of quinin by mouth.

In conclusion, the message I wish to bring to you is that the duty of a physician called to treat a case of malaria does not cease with the relief of clinical symptoms, but that it extends to the disinfection

of the patient and that this can be accomplished by the routine administration of 10 grains quinin daily for adults and proportionate doses for children for a period of eight weeks. If all malaria cases treated by physicians were treated in this way, it would contribute very greatly to the control of the disease.

Q. I have seen cases in which there were two or three parasites in a cell. What about such cases?

DOCTOR BASS: Such cases are simply heavily infected cases and usually present severe clinical symptoms. Most of them recover upon prompt administration of quinin. In fact, all of them do, unless too great damage has already been done before proper quinin treatment can be instituted.

Q. With reference to intravenous injection of quinin, what dose should be given?

DOCTOR BASS: My opinion is that 10 grains of the bimuriate of quinin is the maximum dose that should ever be given intravenously. It should be diluted with at least 20 c.c. of water or salt solution. The dose may be repeated if necessary in an hour or two.

Q. In making a study of tuberculous patients, I have encountered two cases in which there were malaria parasites in the blood, a latent type. These cases were in soldiers who came to Chicago from the South. I apprehend we will get more of these cases in Chicago during the next few months than we have had before. It is worth while being on the lookout for symptoms of malaria in any suspicious cases.

DOCTOR BASS: No doubt there has been considerable distribution and spread of malaria in different parts of the world by soldiers. Fortunately, there was little transmission of malaria in the training camps in this country. Our soldiers were much better off as far as malaria is concerned when they were discharged than when they went into training. In England and France thousands of soldiers have returned infected with malaria while serving in various expeditionary forces, especially in Macedonia. These soldiers have returned to their homes, many of them still malarial carriers, and there is the possibility of rather wide distribution of the disease and perhaps considerable spread.

Q. A man who had clinical manifestations of malaria received only two weeks' treatment. Is that long enough?

DOCTOR BASS: Two weeks' treatment will disinfect a very small per cent. of infected persons. Occasionally one will be disinfected, however, by even a shorter period of treatment than this. The best indication we have that an individual has been disinfected is the duration of effective quinin treatment he has taken. It is impossible to say whether a given individual will be disinfected by two weeks' treatment or not.

DEAN DAVIS: I am sure the audience would like to hear from Dr. Ochsner on this subject.

DR. A. J. OCHSNER: (See Doctor Ochsner's dictation.)

Q. I would like to ask Doctor Bass if he finds any cases in which quinin will not work. I would also like to ask him if he has tried the intermittent method which Doctor Ochsner says works so beautifully. Again, are the cases that are cured harder to reinfect after a course of quinin?

DOCTOR BASS: There are no cases of malaria that can not be cured by proper quinin treatment. There are exceptional cases, however, that do require more treatment than others and more than I have just proposed as a standard routine treatment.

I have not tried in any extensive way the method of treatment proposed by Doctor Ochsner, largely because it has not seemed to me to be practical for routine malaria control work. It may get results in special cases, but even if it does it does not seem to me to have anything to recommend it over less complicated methods.

Cases that have been disinfected by quinin treatment are not more difficult to infect again because of the treatment. It is supposed, and probably is a fact, that previous malaria infection produces a certain amount of immunity or resistance to reinfection. It is not known, however, whether this is ever sufficient to produce absolute immunity and my own opinion is that it is doubtful as to whether it is.

DOCTOR OCHSNER: Supposing you give quinin and the plasmodia come back in five or six days, what then?

DOCTOR BASS: It depends upon the quantity of quinin you give and the duration of treatment. Plasmodia do not come back in so short a time whenever the treatment is proper and sufficient.

DOCTOR OCHSNER: Suppose the parasites return in forty-eight hours?

DOCTOR BASS: Parasites do not reappear forty-eight hours after

sufficiently large doses of quinin to cause their disappearance. They will disappear just as certainly from a given dose of quinin given in one dose during the twenty-four hours as if it was given in many divided doses, as Doctor Ochsner proposes. If only forty-eight hours of treatment is given, parasites will reappear in practically all cases sooner or later.

DOCTOR OCHSNER: How soon after they disappear do they reappear?

DOCTOR BASS: There is great variation and no definite time can be set. It may be a few days, a few weeks, or even a few months. A great deal depends upon the individual and upon the particular variety of parasites and other factors that we do not at present understand.

DOCTOR OCHSNER: But there is absolute uniformity to this method I have outlined because I have examined many, many cases during the last thirteen years and have been unable to find the plasmodia in the blood. I had an assistant superintendent who worked for thirteen years in a malarious section, was never free from malaria, but in ten days after taking the treatment I have mentioned he was free from it and I have not been able to find any plasmodia in his blood since.

DOCTOR BASS: The same thing may occur in cases given very much less treatment than Doctor Ochsner gave. We must not lose sight of the fact that many cases of malaria are relieved by quite a variety of methods of administering quinin. It is not so much the method employed but the amount of quinin and the duration of treatment; the very idea that I wished to emphasize in my remarks. If this is true, then the most convenient and most practical method is the one that should be adopted.

DEAN DAVIS: Is there any evidence that certain strains of malaria become resistant to quinin?

DOCTOR BASS: That idea is responsible for the intermittent quinin treatment, the theory being that daily treatment was more likely to produce resistant strains than intermittent treatment would. There is difference of opinion on this question among the best authorities. So far as I know and believe, there is no conclusive evidence

that malaria parasites may acquire resistance to quinin or become quinin-fast.

Q. What about the idiosyncrasy of people to quinin and what would you do in such cases?

DOCTOR BASS: Usually the thing to do in such cases is to give them quinin if they have malaria. Quinin produces discomfort in a considerable proportion of people who take it, but there are very few in whom the discomfort is as great as the discomfort produced by malaria and there are extremely few, if any, in whom the danger even approaches the danger from malaria. Therefore, it is not difficult to make the choice. It is true that occasionally an individual is made so uncomfortable by quinin that something must be done to reduce the unpleasant effect. Usually taking smaller doses for a few days, gradually increasing while the patient becomes accustomed to the drug, is all that is necessary. The discomfort becomes less and less and soon the full dose can be taken without much, if any, discomfort resulting.

In connection with this question, I would call attention to the fact that in large southern hospitals where a great deal of malaria is treated, the routine treatment usually includes from 30 to 45 grains of quinin daily for the first few days. Nobody is asked whether he can take quinin or not and nobody is found who can not take it. For one person who can not take quinin, there are very many others who fancy they cannot, but who can take it without any considerable inconvenience. I have no personal knowledge of any person who can not take quinin in sufficient quantity to disinfect him of malaria, though I have heard of many.

Q. I would like to ask Doctor Bass as to the rationale of methylene blue or any of the other alkaloids, like cinchona, in the treatment of malaria.

DOCTOR BASS: My opinion is that methylene blue has no specific action in the treatment of malaria and therefore has no place. Many of the cinchona alkaloids have more or less effect on malaria parasites, but none of them are as effective as quinin.

Fowler's solution of arsenic has also been shown to be without specific effect in malaria unless it is given in very large doses, perhaps too large to be practical.

DOCTOR OCHSNER: Are the plasmodia found in any of the domestic animals?

DOCTOR BASS: There are several animals that have parasites that resemble very much the malaria parasites of man. None of them, however, is identical. There is at present some question as to whether a parasite in monkeys resembling the malaria parasite of man may not be very closely related to, if not identical with, the malaria parasite of man.

Q. Why do chills occur practically always in the daytime instead of at night?

DOCTOR BASS: There are a number of theories which offer to answer this question, but the best answer I can give is that we do not know. Perhaps the most feasible theory is that variation in the size of the capillaries at different times may affect the appearance and disappearance of the parasites in the peripheral circulation and perhaps in some way affect their segmentation, which produces the chill and fever. This is a theoretical explanation, however, and I am not at all prepared to endorse it or oppose it.

DR. A. J. OCHSNER: My plantation in Mexico was a veritable hotbed for malaria, and I had employed on it upward of a thousand people, so it was an industrial problem of either going out of business or trying to get rid of malaria, and as a result of that the following plan of treatment of malaria was worked out and has been used since in thousands of cases with very satisfactory results:

TREATMENT OF MALARIA

The following rules are based upon a number of well-known facts:

1. The adult plasmodium of malaria is destroyed in the blood of a patient saturated continuously for forty-eight hours with quinin.
2. The spores of malaria can live indefinitely in the blood of patients without regard to the amount of quinin taken.
3. Spores of malarial plasmodia remain latent in the presence of quinin in the blood and begin to develop only after this drug has been entirely eliminated.
4. These spores require seven days before they can develop into adult spore-bearing plasmodia.

5. Quinin must be absorbed in order to do its work. Hence the importance of the preliminary cathartic, the soup diet, and the hot water taken with the quinin.

6. The blood must remain continuously saturated with quinin. Hence the importance of giving the remedy regularly night and day.

7. The total amount of quinin required is very small.

CONCISE METHOD OF TREATMENT

Follow every step absolutely. Do not miss taking the medicine at regular times at night. Use an alarm clock if necessary. Take the medicine with hot water to insure immediate absorption.

On the evening before commencing the use of quinin, take a cathartic, preferably two ounces of castor oil in beer-foam or ginger ale or root beer; or take five grains of calomel with ten grains of bicarbonate of soda and a Seidlitz powder the following morning.

During the period of treatment it is best to live on hot soups. On the morning following the taking of the cathartic, take two grains of quinin—bisulphate preferred, but the sulphate or the muriate will do—with half a pint of hot water, every two hours, night and day, for two full days and two full nights. Be sure to do this regularly, because missing once or twice will make the treatment useless.

The soup given these patients should be very nourishing, such as rich vegetable soups, milk soups or soups containing an abundance of rice or barley.

Then give absolutely no quinin for six full days and six full nights. On the evening of the sixth day take another cathartic. On the morning of the seventh day begin taking the quinin again, two grains every two hours, night and day, for two full nights and two full days. Then stop and take some simple tonic for a few weeks and keep away from mosquitoes that have bitten others suffering from malaria.

You may use the quinin in solution or in capsules, but in the latter case you must remove the cap from the capsule before swallowing it.

In the interval of six days between the two courses of quinin treatment it is well to take a pill containing one-fiftieth of a grain of arsenious acid one hour before and after each meal, each time with a glass of hot water.

The following prescriptions are usually followed by these patients if one is emphatic in explaining the necessity of obeying absolutely.

In case the quinin disagrees with the patient, it is usually possible to correct this difficulty by giving 2 to 5 grains of sodium bromid in a little hot water before administering each dose of quinin.

Rx.

Quinins Bisulph. in capsul. (aa gr. ij) No. 100.

Sig.

Take one with one-half pint of hot water every two hours, night and day for forty-eight hours on first, second, ninth and tenth day. Remove cap of capsule before taking.

Rx.

Pul arsenious acid (aa gr. 1/50) No. 36.

Sig.

Take one with a cup of hot water an hour before and after each meal on third, fourth, fifth, sixth, seventh and eighth day.

TISSUE NECROSIS AND AUTOLYSIS IN RELATION TO TOXÆMIA: EXPERIMENTAL AND CLINICAL OBSER- VATIONS. TREATMENT BY TISSUE ANTIBODIES *

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New York

INTRODUCTION

IN our treatment and study of disease we are still concerning ourselves too much with the end results and not with the essential cause and beginning of disease. The clinician still treats the end results of a pathological process and the pathologist studies the end result of that process. It may be trite to repeat that if we are going to combat a perverted physiological process successfully we must know the primary cause and this we must seek in the finer molecular changes and reactions that occur between the colloidal particles in the finer cellular reactions—the so-called cellular metabolic changes. That we have not fully grasped the significance of this truth has been brought home to my mind with renewed force by my recent studies of the phenomena of wound shock,^{1, 2, 3, 4} a condition which we have not been able to understand and treat with the success that has followed the modern scientific study of certain other conditions. In the prosecution of my investigations, I have repeated and elaborated many of my former experiments, all of which have been directed, not to the study of end results, but to the early cellular changes in the living subject that have led up to the later and more gross pathological conditions. The evidence of these early changes, as I have demonstrated, are to be found in the architectural alteration in the configuration of the cell and finally in changed relations of groups of cells and in the body fluid, which is shown in the anatomical changes which we have been accustomed too often to consider fundamental. The discovery of a *pathological anatomical* change connected with disease is not the revelation of the cause of that disease, but is merely an indication of the direction to be taken in retracing step by step the sequence of events, the development of the changes observed in the

* Read before the Section on Medicine of the New York Academy of Medicine, May 20, 1919.

living tissue, until we arrive at the true etiology of the disease. This is the biological method which I have consistently adhered to in my experimental work and applied in my practice since 1892, when my first investigations in this line were published,⁵ until the present time, and which have given such uniformity in results that they have convinced me that we stand on the threshold of new and marvellous revelations as to the causation of disease, and possibly these revelations will put in our hands the key to the control of many conditions that hitherto have baffled us.

In order that the reader may get a clearer conception of the significance of our recent work, it may be well to get the trend of thought that has actuated the writer in his work from its inception. Early in the nineties the writer happened to have under his care a patient who was suffering from a severe toxæmia and autointoxication.⁶ The patient gave a history of alcoholism, but had taken no alcohol into his system in three months. He was, therefore, suffering from the effects of a cause which was no longer operative in itself. In studying over the problem of why this should be, the explanation that suggested itself was that the alcohol had initiated a pathological change in the tissues and cells of the body, which they of themselves carried on long after the initial impetus that set those changes in motion was withdrawn. Curiosity to know what the earliest alterations in the tissues were under such circumstances led the writer to make experiments whereby gastritis was caused by various mechanical and chemical agents and the early changes studied in the living object.

A reference to the author's early literature will show that at that time he arrived at the conclusion that it was not irritation alone or bacteria alone that produced the changes that were observed, but that there had to be an alteration in the cell resulting in death of the cell which further disintegrated and itself produced the poison. These dead cells furnished a soil suitable for the development of bacteria, which continued the cellular destructive process. He observed that among the early manifestations of the absorption of the disintegrated cellular products and the disturbance of cellular reactions were changes in the circulation, and in discussing the treatment he stated that it must be directed to the prevention of cell necrosis and the production of antitoxins, to the equalization of the circulation, the prevention of the further formation of toxins and the promotion of

elimination.⁷ It will be seen from a perusal of the author's literature of that period that he recognized the importance of the phenomena of cell necrosis, which we now call cell autolysis, a term that had not been coined at that time. The author comments on "Shock Without Infection," and states that his observations have shown that not only is there a lessened resistance to bacteria and their toxins in the serum of animals that have been in shock, but after a period of shock, the serum assumes toxic (cellular) properties. The injection of the cellular "serum" from an animal which has been in shock into a second animal produced the evidence of shock. These experiments were repeated a number of times and the results were constant in the disturbance of circulation, respiration, lower temperature, and resulting collapse.^{6, 9, 10.}

In 1903, the writer published the results of studies on "Shock Produced by General Anæsthesia, with Relation to Disturbances of the Blood and Gastrointestinal Tract."¹⁰ This study was directed particularly to the consideration of the anæsthetic as a *cellular poison*, with the resulting disturbances of function.* A distinction was noted between the immediate and primary effects and those that were remote or secondary. The conclusion drawn from the details of the experiments made on animals under chloroform and ether was that where shock was produced it did not materially differ from the shock that resulted from trauma. The most constant pathological factor is the failure of circulation, and this is especially expressed in congestion. It was stated at that time that the explanations that were usually given for the complex symptoms that were found in shock from anæsthesia could not be explained by the simple failure of blood pressure and respiration, but there appeared to be some direct *toxic bodies* formed which were found in the blood serum.

Believing that cell necrosis played a large part in the production of pathological processes, I conducted experiments producing cell necrosis by both physical and chemical means. Various chemical agents known to produce injury and death to the cell were applied to the tissues. Various grades of injury ending in necrosis were

* The experiments at that time showed that cell necrosis evolved a product in the nature of the products of digestion found in the stomach which, on injection into animals, was found to produce the same phenomena of shock and death in the animal.^{1, 22}

obtained by the prolonged application to the tissues of chromic acid, silver nitrate and tannic acid. The evidence of cell necrosis was determined by microscopic examination *in vivo*, by studying the products of necrosis removed from the stomach wall by means of the gyromele.^{7, 11}

The physical means of producing necrosis of the cells was by exposure of the viscera to the air, which acted as a burn would act to the skin and produced results identical to those following mechanical injury, such as the incision or bruising of the tissues. Prolonged exposure with rough manipulation increased the degree of cell injury and resulted in more extensive cell necrosis, producing a profound shock and death.⁶

An experiment typical of those carried on at that time was as follows:¹²

Two dogs, brothers.

One was placed in shock by exposure of the abdomen to air and manipulation for one hour and fifty-five minutes. The animal died.

Peritoneal fluid scraped from the peritoneal and lung cavities of this dog was taken and injected into the second dog, with the result that 10 c.c. caused a state of collapse.

Rabbits were also injected with this "serum," with the result that convulsions were caused similar to those produced by snake poison.*

The shock phenomena was primarily the congestion and stasis of the splanchnic circulation, and this was the direct cause of death. That the cell necrosis and disintegration of the cell was the direct cause of the shock was proven by the fact that the injection of the products of digestion from the stomach into animals produced shock and death of the animals with the identical splanchnic congestion and all the other evidences of shock that were noted in the animal from which the material for the injection was obtained. The injury to the tissues was produced by exposure of the viscera to the air and the mechanical trauma causing the cell necrosis and absorption of toxic products which caused death.^{1, 2, 3, 4.}

Cases were reported¹³ in which the toxin of bowel obstruction,

* In these early experiments the term "serum" is used in rather a loose way as indicating the exudate containing necrosed cells. Where muscle was used it was called "extract" and *bouillon* toxins.¹⁴

acute gastritis, pyloric obstruction, by absorption gave the identical conditions observed in shock caused by mechanical injury, or those following surgical operation. The opened abdomen in many of the cases gave opportunity to demonstrate that the shock symptoms and the splanchnic congestion and stasis were the same in each case; that is, the color in each instance would be the same at the same stage of shock. That the toxins producing the shock were of the nature of the *products of digestion* was proven by the fact that the stomach contents when injected into the peritoneal cavity of other animals *produced the symptoms of shock*. Recovering the fluid from each of these cavities containing the disintegrated cells and serum and again injecting it into other animals resulted in the death of these animals from shock, which was the same as that manifested by the animals in which it had been induced by trauma or surgical operation, or the injection of products of digestion taken from the stomach.

The *particular* histological lesion that occurred in shock was found in the submucous tissue of the intestines and portal and periportal zone of the liver. This was especially noted when intravenous injections of stomach contents were made in animals. Not only was it shown that in both animal experiments and clinical study of a large number of cases injury of the cell caused death of the cell and a resulting poison that produced the phenomena of shock, but also that if bacteria were introduced while this process was progressing the microorganisms were able to progress very rapidly. In this connection we also demonstrated that germs showing no pathogenicity, such as the *Staphylococcus albus* and the *B. coli*, when injected alone into animals did not cause death, but that if disintegration had been previously begun in the tissues, the addition of the bacteria caused a further and more rapid disintegration of the body cells, because of the increased dosage of the products of cell disintegration.^{9, 10} This was manifested by an increase in the shock symptoms and a more rapidly fatal termination. I would emphasize this point, that it is the multiplication of germs in the already shocked or injured tissue which increases the cell degeneration and production of toxins, although such bacteria may not cause ill effects when injected into the blood. In other words, although a "toxin" is produced by microorganisms, it is not capable of causing death directly, not until

necrosis of the cells is first initiated. The dead cell of the body is the direct agent that produces pathology.

The effect of the poison that causes cell *necrosis* is further shown by administering chloroform and ether to animals for varying periods of time (from one to six or eight hours). Shock and death are the result, not of the specific toxin of the chloroform or ether, but the direct effect of the toxins produced by the action of the chloroform or ether upon the cells of the body.

Non-pathogenic germs, such as *Staphylococcus albus* and *B. coli*, were equally prompt in their toxic effects when injected after shock produced by anaesthesia. When injected into normal animals used as controls they gave negative results.

That the anaesthesia produced by ether or chloroform is not the direct cause of shock and death is shown by the following protocol:¹⁴

1. The injection of fresh cells taken from an animal and immediately injected caused no symptoms.

2. Adding chloroform and then injecting the products of the disintegration caused by the chloroform or ether caused death.

3. The injection of 2 c.c. of chloroform in a cat causes cell necrosis and death. Removal of the disintegrated tissue and injection of this disintegrated tissue into an animal causes death of the animal.

Control.—Two and one-half c.c. of chloroform administered alone did not cause death unless it first destroyed the tissue cells.

THE ROLE OF BACTERIA

It was shown in my experiments that combining autolyzing cells with *B. coli* increased the rate of autolysis of the cells and toxins were formed which produced death from shock when injected into animals. Blood serum from the femoral vein mixed with the *B. coli* did not cause death, nor did the *B. coli* when introduced alone.^{9, 10, 40}

The conclusions from my earlier work on shock phenomena, by whatever methods produced, were that the facts brought out established a most important point in the pathology of shock, namely, that the alteration of the tissue cells and then of the blood were the primary factors concerned in initiating shock.

The evidence that the occurrence of blood changes is also primary and coincides with the cellular changes in shock is demonstrated in the congestion of the abdominal viscera in shock.¹⁵ "We cannot ap-

parently produce profound shock without the occurrence of congestion, and the reduction of such shock does not occur until we reduce the congestion and distribute the blood over the surface."

The study of shock phenomena is essential to an understanding of what occurs in many other diseases in which the necrosis of the cells and the absorption of toxic products initiates the pathogenesis of the whole disease process. The toxins of cell disintegration not only cause general disease, but frequently determine specificity in disease. Thus, I injected tissue from the stomach mucous membrane and also from the muscle tissue, and found that it caused atony of the stomach and intestines.¹⁶

In 1893, I made experiments in the production of gastritis by local cellular injury, causing necrosis of the cells, and then introduced bacteria, and finally succeeded in establishing a chronic gastritis.^{7, 11} As the condition progressed there resulted from the absorption by the walls of the stomach a lack of motor power of the stomach itself, which shows that the cells by the absorption of the products of cell necrosis acted as a poison on the muscle tissue. These products injected into animals produced atony and dilatation of the stomach.¹⁷

PRODUCTION OF PEPTIC ULCER

Ulcer of the stomach was produced by the writer, not *directly* by feeding animals with bacteria and beef extract, but indirectly. The feeding of animals with large cultures of the bacterian and meat extractives resulted in partial starvation, which always follows deficiency feeding.

Experiments.—To make up for this want of nutrition the tissues undergo self-disintegration, or to employ the expression used at that time, "cytolysis and autolysis."^{22, 22} The bacteria or the extractive were not the *toxic* factor—it was the product of cell necrosis. This was shown by the fact that no bacilli were found in the tissues or in the blood. It was later found that bacteria from the intestines might diffuse into the tissues, and while being themselves destroyed in the submucous coat, they caused the cells to undergo necrosis, owing to the loss of antibodies, and finally an autolysis that created the conditions suitable for the development of ulcer.^{23, 22}

Stewart and West²³ considered that this ulcer-forming status obtained in my experiments might be due to *acidosis*, which led to the

formation of ulcer, but the experimental evidence points to the acidosis as a secondary phenomenon resulting from cell necrosis. Cell necrosis always causes further cell necrosis unless checked by the normal antibodies. If cell necrosis is excessive or continued over a long period, then the ulcer status is established.

FERMENTS IN CELLS—BACTERIA

The cell is well equipped with ferments (trypsin, erepsin, pepsin, etc.), which act in an orderly specific manner on the food products and other substances brought to the cell by the blood, and when the cell dies it is itself digested by its own ferments. This is termed autolysis, and Dernby²⁴ defines it as follows: "By autolysis we mean the no-bacterial post-mortem self-decomposition of animal or plant tissues." Salowski,²⁵ in 1891, first recognized the phenomenon as due to enzymatic action and called it "autodigestion." The name "autolysis" was introduced later by Jacobi, in 1900.²⁶ Dernby²⁴ concludes that "considering that there are enzymes in the tissues of both peptic and tryptic nature, and considering also the hydrogen-ion concentration of the medium and its eventual changes, most of the autolytic reactions can be explained without the assumption of hypothetical agents, such as 'proenzymes,' 'activators,' or 'anti-enzymes.'" Rice,²⁷ in 1915, shows the rôle of erepsin in tissue autolysis, especially in tissues of the intestinal tract.

Cellular tryptase and peptase are intercellular enzymes which Effront²⁸, Robertson²⁹ and others have studied showing these proteolytic ferments belong to the same group as the digestive ferments with which we are familiar in the process of ordinary digestion.

Cellular autolysis (tissue self-digestion) seems to be but a continuation of the intracellular vital process. It has been shown by Sykes and Myer³⁰ that during a prolonged fast the normal content of amino acids is maintained in the blood and muscles through autolysis of the muscle proteins. This mechanism of safety during starvation shows that the blood always contains a suitable pabulum to support the nutrition of the nervous system, internal organs, and heart, even at the expense of great wastage of the skeletal muscles, as McCollum and Simonds³¹ have maintained, but as they conclude, life is cut short if the experimental conditions imposed are sufficiently rigid and the death of the animal is preceded by toxic symptoms and loss

of muscle control. In starving by more gradual methods the toxic condition is less pronounced.

Deficiency disease I found had a similar pathogenesis. Feeding animals on extracts of beef and *B. coli* finally gave the pathology in the form of degeneration changes in every organ of the body. I called attention to the autolysis of the tissues produced in these experiments which induced the formation of typical peptic ulcers. The deficiency of the hypothetical vitamins is in reality an autolytic process in which the body tissues are self-digested to supply the deficiency, and during this process the body is poisoned by the products of tissue autolysis, due to autocytolysis (Turck).³²

Feeding monkeys heated fat in connection with ordinary food caused deficiency in utilization of food, resulting in autolysis, with atrophy of all the animals and death in from twenty-eight to sixty-one days.³³

Feeding animals with extracts of beef and large bouillon cultures of *B. coli* produced a food deficiency disease in all the animals. Many animals died from ulcer with hemorrhage and perforation. Degenerative changes were found in all the organs of the body.^{32, 31}

Schryver³⁴ shows that in starving animals the liver autolyzed more rapidly than that of normally fed animals. According to Schryver, the liberation and activation of the autolyzing enzymes in the cells during life is a normal mechanism, whose function is to protect the organism against starvation. Simonds³⁵ concludes that these enzymes come into action whenever the energy needs of metabolism are not satisfied by the food ingested.

By reason of the speed with which the polypeptids are formed, these products of autolysis may become extremely toxic. I observed in 1896 (1897)^{17, 36} that the injection into animals of polypeptids obtained from the stomach cavity was followed by loss of muscle control, "dragging the hind legs as in snake poisoning," followed by deaths, the same as occurs in experimental starvation when the conditions imposed are sufficiently rigid. It is immaterial by what method we produce autolysis, whether by cutting off the circulation or other injury of the tissues, if the conditions imposed allow for speed of ferment action on the tissue cells, the absorption of the products will cause the symptoms of poisoning and death.

The interruption of the blood supply to the liver, I found (1893),³⁷ on tying off the portal vein, caused death of the animal,

but partial tying off of the pylorus did not result in death. Complete occlusion of the pylorus resulted in death of the animal with toxic symptoms of shock. Ligation of the vessels of the lesser curvature of the stomach did not cause death, but caused serious congestion as in shock, with hemorrhagic spots and erosions in the stomach (1897).³⁷ Similar results have recently been obtained by Gunderman (1913). Jacobi (1900),³⁸ has shown that the liver lobes whose blood supply has been interrupted undergo autolysis. Senn (1898)³⁹ has demonstrated that the symptoms of intestinal obstruction, necrosis and death are due to interruption of the circulation. The necrosed and gangrenous tissue product is the poison (given off by the self-digested tissue) that caused symptoms and death, and not the products from the lumen of the obstructed gut. My experiments show the early pathological changes that occur after intestinal obstruction. The injury to the wall of the gut by obstruction and the blood stasis created by mechanical interference with the circulation always resulted in death of the cells at and near the point of obstruction.³³ When this occurred in the higher segments of the duodenum and jejunum, where the supply of ferments is richer than in the lower segments, the autolysis is more prompt and death occurs more rapidly. The absorption of the products of the more rapid autolysis shows the early pathological changes in the liver and lungs. Later the toxic effects may be observed in the kidney and other organs.

By adding the ferment products from the submucosa of the intestinal wall to the tissue cells of the animal, and then incubating for half an hour, it was found that toxins rapidly formed.³³ This produced immediate death on intravenous injection into animals of the same species. Control animals rendered immune by anti-serum did not die. In animals rendered immune, the ligation of high loops of intestine did not cause immediate death as in the unimmune animals. When death did occur in the immune animals with high obstruction, it was the same as in slow starvation, or in animals in which there was obstruction of the lower segments.

SUMMARY

In summing up, it may be said that my first conception was that diseased conditions of toxic character persisted long after the original causative factor had been removed, and that they were not due to

mechanical causes, but to toxic causes, as was suggested by the case of poisoning by alcohol, in which long after the alcohol had passed out of the system the secondary toxic effects were demonstrable. It was shown that inflammation of the mucous membrane and necrosis was produced by tannic acid, silver nitrate, etc., which resulted in exfoliated substances composed of dead cells. Then bacteria introduced found the bed or soil suitable and further continued the necrosis begun by the tannic acid or silver nitrate. The absorption of these products' necrosed cells produced an effect in the tissues beneath, even to the muscular layer and caused not only the pathological picture of gastritis, but also the failure of the glandular function, and finally impaired the muscular coat controlling the motor power of the stomach. The primary stages produced at first increased hydrochloric acid and ferments, and later loss of these secretions, until in the later stages we found atrophy of the stomach wall.

Circulation.—We found that not alone did the mucous membrane suffer as the result of the necrosis, but that during the early stages the circulation of the part was materially altered. The congestion was not passive, but there was a stasis of the blood.

Our experiments showed that animals sensitized with an emulsion of tissue from the *zona transformans* and then receiving an injection of homologous tissue exhibited symptoms of acute venous dilatation of the splanchnic vessels, and this was apparently the cause of the sudden collapse and death.³³ To determine the effect of the anaphylactic reaction on the splanchnic vessels, the abdomen was opened and the vessels examined under the lens at the moment of the injection of the intestinal bacteria and submucous material. A prompt and immediate dilatation of the splanchnic veins was recorded, with a corresponding contraction of the arteries in each animal under observation, and collapse and death followed immediately after the injection. In those animals in which there was a delay in collapse and death there was also retardation in the intestinal venous stasis. Sublethal doses caused proportionately slight venous stasis followed by recovery.

GASTRO-INTESTINAL ATONY AND DILATATION

Muscle Coat.—Microscopical histological examinations at intervals in the progress of experimentally produced diseases show the

changes that occurred step by step. It was shown that the muscular apparatus soon suffered, there being at first a slightly increased activity, which was followed by loss of motor power, atony and dilatation. After the motor power of the stomach and intestines had failed and changes were induced in the circulation (blood stasis), we were confronted with the problem offered by general acute splanchnic stasis. This is observed in all its classical manifestations in *shock*, which also is accompanied with a definite syndrome that follows immediately, *e. g.*, fall in temperature, low blood pressure, and metabolic failure.^{40, 41, 42, 48}

The conditions that cause cell necrosis and produce "shock" are incisions in operations, exposure and handling of the abdominal viscera, and the conditions that follow indicate that poison is formed in the *nature* of partly digested material, such as is found in the stomach. The injection of the stomach material or that obtained from the dilated stomach caused shock, with the production of a shock toxin (albuminoses). The injection of necrosed cells with the serum fluid from both peritoneal and pleural cavities produced on injection identical shock phenomena, being the identical effect produced by shock when the viscera were exposed, and also the same as when the stomach albuminoses were injected. In all these instances the character of the blood stasis was the same, *e. g.*, splanchnic stasis.

✧ *Order of Pathological Events.*—The order of events described was, therefore, first, the injury that produced death of the cell; second, the formation of a poison made up of the fragments of the disintegrated necrosed cells; third, the absorption of the poison of necrosed cells, and, fourth, immediate stoppage or stasis of the splanchnic circulation, affecting the venous capillaries. The proof of this lay in the microscopical examination after the injury and the fact that injecting these injured cells into a normal animal produced identically the same condition; that is, the splanchnic stasis, all of which could be watched and studied as it progressed with a magnifying lens within the abdominal cavity. Histological sections made *in vivo*, frozen and stained, show that the stomach, intestines, liver and lungs are primarily affected. Under severe conditions, other viscera were involved in this *stasis* in the finer venules, the blood backing up gradually into the larger veins or diffusing into the tissues.

When seeking for the nature of the products which resulted from

the injection of contents from a dilated stomach into a normal animal which caused these same phenomena of sudden stasis in the splanchnic vessels, we carried out experiments in which we employed fractional coagulation by heat at various temperatures. By filtering the fluid through a Pasteur filter¹⁷, it was found that one part of the product was more toxic than another, but that the nature of this toxin was altered by the presence of peptones, and that peptones injected produced a similar phenomenon. A comparison of the products of disintegrated cells and products of digestion from the stomach peptones acted like a snake poison on some of the animals. Comparison made with other poisons, such as diphtheria toxin, showed the difference in the effect.⁴³ The stomach peptones when injected into animals produce a congestion in the splanchnic vessels of the intestines and in the portal and periportal zone of the liver. This was similar to the conditions found in shock by whatever manner it was produced. (It was found that the diphtheria products had a more direct digestive effect on the cells, and that they later created a necrotic cellular poison.) It was found that atony of the abdominal viscera occurred in connection with splanchnic stasis. All the evidence goes to show that there are many other diseased conditions associated with this same phenomena.

It was early found that the only agent that altered the phenomena of shock, however it had been produced, was *heat* applied directly to the splanchnic area. The heat produced some physical effect which caused an alteration in the biological process. Heat applied within the abdominal area after or during the exposure of the viscera, bruising of the tissues, etc. (increasing cell necrosis), caused a peculiar effect not only on the blood stasis, but upon the very *poison* that was instrumental in producing the splanchnic stasis. That heat altered the poisons as they were formed is stated in my protocols.⁴⁴ In other words, we demonstrated that the condition which followed and was associated with the poisoning, from whatever cause produced, was reversed by heat.

☛ *The Character of the Poison.*—It is, therefore, demonstrated that the product of cell necrosis produces a secondary effect, which we term blood stasis. The results of cell necrosis may be summed up then as: (1) Gland inflammation; (2) muscle insufficiency; (3) acute shock, with splanchnic stasis as the fundamental phenomena, and (4) sub-acute or chronic splanchnic blood stasis.

General Pathological Condition.—It is now seen that the writer's original conception of disease remains practically the same; namely, the *congestion* of the splanchnic areas is the chief pathological condition. We have learned more exactly why such congestion is present and what it signifies in producing disease and death. The mechanism of this change is apparently a specific reaction. When Jenner observed, in 1798, that congestion occurred in a woman's arm, when injected with "variola matter," who had had cowpox thirty-one years before, he opened the field of anaphylaxis. But this reaction depends upon previous *sensitization* of the protein, which Vaughan and Wheeler show is identical to the "split proteins," and is not specific. In our own work it has been repeatedly shown that *species reaction* is specific—that the "homo-tissue" products are the primary cause of disease and death, and that no previous sensitization is required to initiate their activity. To put the matter plainly, man is already sensitized to his own disintegrating tissue products, which, when absorbed from any injured part of the body result in disease and death.

As Zinsser says, the cytolytic antibodies may be quite distinct from the albumino-lytic amboceptor which is developed against protein antigen.

Experimental and clinical work presented by the writer has shown that many diseases, both acute and chronic, are the direct result of poisons generated by the dead tissue cells of the body. The nature of the poison was shown to be due to the absorption of the product of the cellular disintegration, albumoses or "peptones," which was specific to the species. The dramatic and mysterious phenomena of shock was easily recognized by the writer as the result of a poison generated by the body tissue cells. Other pathological conditions were also found to be due to the same cause, modified by the various degrees of toxicity and immunity reactions. A grouping of the writer's literature on the subject may be shown by the following outline:

The general principles of the writer's research from the beginning of the experimental work in cell necrosis and the resulting pathogeneses and pathology dates from 1893, of which the following outline summarizes the numerous and complicated experimental protocols:

I. Etiology and Pathogenesis.

- (a) Any factor, mechanical, physical or chemical, that causes death of the cell is the starting point.
- (b) Conditions that permit rapid disintegration of the cells and the formation of toxins from these products are etiological and pathogenetic factors.

II. The Special and General Pathology was clearly defined as a primary stasis of the splanchnic circulation.

III. The symptoms occurring as a result of splanchnic congestion were varied. If symptoms were immediate there was a fall in blood pressure and temperature, chills, trembling and other well-recognized secondary phenomena. Reactions later caused a reversal of these symptoms, and treatment was directed to the induction of these reactions by heat stimulation, the stimulation of antibodies by anti-sera, etc.

Treatment. Indications and Methods to Cause Prompt reaction.

—Heat was found to be not only a physiological but a biological method. It must be applied not only to the surface, but directly within the splanchnic area. A small hot-water rubber sac attached to a stomach tube was introduced into the stomach (1896)⁴⁶ “to affect the portal vessels” in the liver and the mesenteric vessels, as well as the gastric vessels, for it was forced upon the writer as a dogma (1897)⁶ “That we cannot apparently experimentally produce profound shock without the occurrence of congestion, and that the reduction of such shock does not occur until we reduce the congestion and distribute blood over the surface.” Reed (1901) applied this principle in his practical work.⁴⁷

The immunity produced by heat applied within the splanchnic area was also observed as a biological method, which may be seen by reference to the writer's extensive literature on shock.

- ✓ Immune bodies are formed in all shock, but when death occurs it is because these immune bodies are not sufficient. Certain immune bodies, such as agglutinins, precipitins and hæmolysins, were observed in 1903.¹⁰ Opsinins are formed by dead bacilli and various antibodies produced by foreign protein injections. The antigen of autolyzed tissue and the transference of homologous sera^{10, 16, 28} lead

to the production of a specific antitoxin which has yielded the most brilliant results of any remedy yet employed.

The antigen used to produce this specific anti-sera is obtained by using dead human cells autolyzed under aseptic conditions and injected into an animal at regular intervals until a high titre is obtained. The successful specific results obtained by the use of this anti-serum in animals and in the human species is corroborative evidence of the rôle of cell necrosis in the causation of disease.

With this conception of the etiology and pathogenesis of disease, I have treated a large number of various groups of diseases, basing my treatment upon exact findings of altered function in physiology, pathology and biological chemistry, upon histological studies made in animals and the pathological anatomical findings in post-mortem cases. In all of this work I have found that my practical experience parallels the experimental work and gives convincing support to my belief in the correctness of my conception of the etiology of disease. Many cases have been followed to the operating table and specimens secured in which the architectural cellular pathology gave final evidence of the correctness of our experimental and clinical studies:

The classification of diseases according to textbook methods is misleading, because what one sees is not the disease, and the anatomical changes observed often represent only the final reaction in a long series of pathological changes. To make a clinical division of the cases for the identification of various conditions and stages the following classes of cases are here reported:

1. General:

So-called altered metabolism:

Gout,
Rheumatism,
Kidney.

2. Cardio-vascular:

High and low blood pressure,
Cardiac dilatation,
Arrhythmia.

3. Alimentary Tract:

Atony of the stomach and intestines,

Secretory disturbances. Peptic ulcer.

4. *Surgical Cases:*

Emergency cases,

Infectious cases,

Surgical prophylaxis and after treatment.

Because a patient has been operated upon necessitating incision wounds, exposure of the viscera to the air, and anæsthesia, there are shock conditions that linger long afterward. If, in the beginning, there is a lack of resistance associated with the condition for which the operation was performed, we must expect the operative risk to be much increased. The indications for treatment by immunizing methods, are, therefore, imperative.

In the treatment of the following class of cases I have followed the methods of colonic and stomach lavage and dietetic and other remedies described in my literature in accordance with the indications of the individual case, but I am convinced that the *specific treatment* by the use of the antitoxin serum for producing active and passive immunity is the only exact and effective treatment in these cases. The dosage of this serum is from 1 to 10 c.c. The active immunity was more quickly established by one or two separate injections of chloroform ($\frac{1}{4}$ c.c. at each point of injection). These injections were usually made subcutaneously in the lumbar region.

The following cases are selected because they had undergone various forms of treatment, all of which had failed, so that we could feel that the results obtained were evidently the direct and prompt response exclusively to the antitoxin serum treatment.

Specific Immunizing Serum.—The immunizing serum employed is made by the intravenous injection of human autolyzed tissue into horses. Small injections are made into the jugular vein of the horse two or three times weekly for six months until a high titer of antibodies is created. These antibodies are tested against human cell products *in vitro* and further biologically tested. Comparisons are made with animals immunized with goat "antitoxin" serum. This is made by the injection of the goat with animal's autolyzed tissue specific to the animal used. Various portions of the human body are used for the antigen. The heart seems to give the most polyvalent effect. Gastro-intestinal tissue in ulcer work and respiratory tissue,

including lung tissue, are used for antigen for producing a serum for immunization in respiratory diseases.

Dosage and Number of Injections.—In the beginning of the course of treatment we inject 3 to 4 minims of chloroform at each selected point. This is followed by the separate injection of the specific immunizing serum, using 3 to 50 c.c., according to the demands of the condition. The number of injections is also a matter of the physician's judgment. In many acute conditions one or two large injections are all that is required. In subacute and chronic conditions several injections are used, about ten days apart. After the initial injection of chloroform it may be later discontinued and the antitoxin injected alone. Slight redness will occur and sometimes the usual rash, but this has not been so constant as in the use of ordinary serum injections. We have seen no untoward results and none have been reported. All testify that it is practically harmless.

CASE REPORTS

CASE I.—*Peptic Ulcer.*—Miss R. S., age thirty years. This patient complained for five years of pain in the pit of the stomach radiating to the back, and increased by the ingestion of food. The pain awakened her at midnight and continued until the rising hour. She had hyperchlorhydria, blood in the stools and occasionally in the stomach. In fact, she gave the classical ulcer history. Several attempts at dietetic "cures" had been made without lasting benefit. There were acne and furuncles of the advanced type on the face, back and breast. For this condition she had had the usual treatment without permanent results. She had been treated by foreign protein injections at intervals for one year. This had resulted in some symptomatic benefit, but no marked change in the ulcer status or the acne and furunculosis. Operation was refused. The diagnosis of peptic ulcer had been made and confirmed by the X-ray.

She was injected with 5 c.c. of "antitoxin" serum, with two $\frac{1}{4}$ c.c. chloroform injections. This was repeated in ten days, and was followed by some reaction.

Result.—There was a disappearance of all pain and other signs of ulcer. The acne and furunculosis disappeared. She has resumed her occupation and is without symptoms.

One year after she shows no return of symptoms and is perfectly well. All the clinical findings indicate a permanent result.

CASE II.—Chronic Ulcer Following Burns.—Mr. F. K., age forty-eight years. This patient was a moulder who, fifteen years ago, suffered severe multiple burns from molten metal. These burns were followed by pains in the pit of the stomach and vomiting. The vomitus contained blood, and blood was also found in the stools. The patient became anæmic and incapacitated for work. All forms of ulcer treatment were given with varying results. After three months in bed, with dietetic treatment at intervals, it became possible for the patient to resume work, only to have the ulcer symptoms and clinical findings repeated soon after leaving the hospital.

The patient was repeatedly warned of the danger of perforation or of cancer appearing at the site of the ulcer, but refused operation.

Examination showed the typical form of pyloric ulcer.

Eight injections of the antitoxin serum were administered over a period of two months. The beneficial effect was noted from the first and continued until cure was complete.

Six months after the cessation of treatment there had been no signs or symptoms of ulcer. There is no anæmia and the patient has gained in weight. He is able to continue his occupation as a moulder without fatigue, pain or any untoward symptoms.

CASE III.—Duodenal Ulcer and Ulcer of the Bladder.—The following is the summary of a report made by the attending physician, Maj. Voss, on a case treated with the serum for ulcer of the stomach and bladder:

The patient was a dressmaker. She was suffering from hemorrhages from the stomach and bladder. She had been under treatment by the usual methods, but continued to grow worse until she became a chronic invalid, suffering such severe pain that she was unable to work.

She was given two injections of the antitoxin serum, which resulted in a strong reaction followed by immediate improvement and final cure. To quote Maj. Voss, the exact words of the report: "This case, Mme. D., treated with your serum, shows remarkable results. The hemorrhage from the stomach and the blood in the stools has entirely ceased. The stools, which were black and tarry, show no signs of blood. The

symptoms of ulcer in the bladder, blood in the urine, irritability of the bladder, etc., disappeared at the same time that the blood in the stools cleared up. The patient has gained twelve pounds since the treatment was begun. This is the most remarkable cure I have ever witnessed. The patient has been able to resume her occupation of dressmaking."

CASE IV.—*Gastric Ulcer.*—Miss C. M. R., twenty-seven years of age. This patient complained of the following symptoms: Pain on pressure over the gastric region, penetrating to the back; pain immediately after meals, increasing in severity two hours after taking food; cramps; nausea after every meal; retention in the stomach. Raisins and rice remained in the stomach twenty-four hours. The patient complained of a "sour stomach"; hyperchlorhydria was always present and was relieved for a time by bicarbonate of soda. The patient was anæmic, the hæmoglobin being 70 per cent. In addition to these symptoms the patient complained of general weakness and inability to work, of headache, depression, insomnia and menstrual irregularity.

The general examination, including the X-ray examination, confirmed the diagnosis of peptic ulcer. The usual ulcer treatment—rest, diet, alkalies, arsenic, iron, injection of vaccines and other foreign proteins—failed to bring relief. Finally operation was decided upon.

In order to prepare this patient for operation, as she was not a good operative risk, she was given the heart antitoxin serum treatment. Four injections of 3 c.c. each of the heart antitoxin serum were administered over a period of six weeks.

Results.—Improvement was manifested from the beginning. The pain, nausea, gastric retention, acidity and insomnia disappeared. The menstrual pain and headaches, as well as the mental depression, cleared up, and the patient resumed her work without any distress, and has remained well since, with no return of symptoms. She has no evidence of ulcer and the hæmoglobin is 85. The surgeon has discharged the case, since there is no indication for operation.

CASE V.—*Shell Shock.*—Mr. I. B., fifty-nine years of age. The following is the typical history of so-called shell shock in the patient's own words:

"I was on the British battle front in September, 1917, and one

morning as we were ascending the gradual slope of the Vimy Ridge, with the British trenches about one-third of a mile ahead, a group of five being in our party, I suddenly observed a Boche airplane in the sky over the German lines. We thought nothing of it and proceeded on our way. We were abruptly stopped by the sound of a great shell coming toward us in the heavens. This shell probably carried a vacuum as big as a freight car, and as it neared us many sounds proceeded from it, the whole resembling an express train travelling at full speed very close to us, with the roar of wheels and whistle. The shell landed about eighty yards behind us and directly where our feet had been a few seconds before. We had not recovered from our *astonishment* when another shell came and landed with a terrific *detonation* in about the place of the other. Our guide, a British officer, said, 'Some German battery must be gunning us, let us get under cover of this half-ruined villa.' So we stepped into the open doorway of a shot-up villa opposite which we had been standing, and while there these great shells continued to come over. The nervous tension of the moment was rather extreme, and the sound of the approaching shell suggested to one that it was likely to land on his own person. I felt a sharp pain in my eye after the first detonation, which turned out to have been caused by a minute fragment of steel which had imbedded itself in the cornea and which was removed at a general hospital in Etape.

"I had other experiences, notably one on Hill 304, although no shell landed nearer me than 100 yards or so.

"On my return home, my friends observed that I had lost flesh, and I found on weighing that this was true; I had lost, if I remember rightly, about ten pounds. This loss of flesh continued until my weight diminished by twenty-two pounds."

This patient states that he had always been well before the "shock" experience. He now suffered from indigestion, headaches, depression, incapacity for work, great nervousness, sciatica, frequent urination and irritability of the bladder. Ureteral examination showed pus, especially from the right kidney. *B. coli* had apparently diffused from the colon. Physical examination was negative except for functional atony of the alimentary tract.

Five injections of the antitoxin serum were administered over a period of two months.

Result.—All symptoms disappeared, the urine cleared up and the digestive apparatus resumed its functions. The patient regained his former weight. He reported eight months later and the restored status seems permanent.

CASE VI.—Cardiac Case.—The following two cases show interesting symptomatic results obtained in advanced chronic myocarditis.

The first patient, Mr. G. S., aged sixty-five years, complained of great dyspnoea and oedema of both legs up to the thighs.

Physical examination shows the typical myocarditis, with extrasystole, systolic murmur, etc. The diagnosis was confirmed by the electro-cardiograph tracings. There did not seem to be much hope of controlling the advancing oedema and dyspnoea.

After four injections of the antitoxin serum the oedema, dyspnoea and general weakness gradually disappeared and excellent functional compensation was restored. No other medical treatment was given. The patient is able to do light work and suffers no apparent inconvenience. With careful general treatment he may live some time.

CASE VII.—Mrs. O'B., laundress, sixty years of age. This patient complained of great weakness, shortness of breath, swelling of the legs, feet and hands, and cardiac dyspnoea. She had been in bed under a physician's care for myocarditis and dilatation of the heart. The doctor gave a bad prognosis, believing the woman was fatally ill.

Physical examination showed that simply the exertion of rising caused great dyspnoea. The functional test showed dilatation of the heart, murmur, regurgitation and dyspnoea. There was arrhythmia and a weak muscular action.

On April 1, 2 c.c. of antitoxin serum was injected, and on April 2 and 27, 1 c.c. of the serum was administered. Gradually all the symptoms have disappeared, the heart shows regular action and all the oedema has disappeared.

On May 3, the patient felt well and had resumed her work as laundress. She states that she can work with comfort. On this date she was given another injection of serum.

June 1, the patient reports that she has been doing housecleaning with ease and comfort. She suffers from no cardiac dyspnoea. There is no murmur, no dilatation and no oedema.

July 1. Examination shows no return of symptoms and the patient works without interruption.

CASE VIII.—*Neuralgia*.—Mrs. B., aged sixty years. This patient had been sick for three and one-half years before coming under treatment here. She had had an acute attack of "flu," followed by exceedingly severe neuralgia, affecting more particularly her head. She was completely prostrated by the excessive pain.

December, 1918, she received three injections of chloroform and the antitoxin serum at three different times.

Result.—There was an almost immediate improvement and a gradual recovery, so that within a week she was in almost her normal health. Two weeks later she looked and felt like a different person.

On January 25 she was apparently in her normal health. The dentist stated that she had pyorrhœa and examination of the ears shows the scar of an old perforation. This patient states that before taking this treatment she was greatly troubled with intestinal gas, but she now notices that this trouble is no longer present to any extent.

Examination in June, 1919, shows the patient in good condition.

CASE IX.—*Arthritis*.—Mr. H. G. This patient was suffering from an incipient stage of arthritis deformans. His chief complaint was severe upper cervical lameness and stiffness, with inability to move his upper extremities without pain. The seventh cervical to the third dorsal spines were very painful to the touch. The lower extremities were swollen and painful. The patient was unable to use his hands in writing. He suffered from severe burning sensations of both feet, with inability to walk. He also suffered from headaches and physical exhaustion. He had been treated by different physicians and in several sanatoriums, springs, etc.

Under serum injections the patient's condition improved from day to day, and within three weeks after the first injection his condition was so markedly improved that he was able to go about his daily work unattended. He is now spending his time in the country, writing in the morning and doing considerable garden work in the afternoon.

The patient made the statement, on February 22, 1919, before going South on a three weeks' lecture tour for the Colleges for the Advancement of Literature, that his whole system and general health had shown a radical change. He later reported that he delivered four lectures in Cleveland in three days. On another occasion he lectured four hours in one day, and again he spoke five times in three days.

He can eat anything and walks long distances. He says, "All my friends wonder at my marvelous cure. I am full of activity every hour of the day, up to 12 o'clock at night." He has organized a Roosevelt Committee and does a great deal of work writing letters, etc., conducting all the correspondence for his lecture bureau. On his return from his lecture tour he was able to perform a large amount of physical and mental labor, showing a most remarkable endurance.

CASE X.—Arteriosclerosis. High Blood Pressure.—This patient, T. B., fifty-six years of age, feels flushed, and suffers from a sensation of dullness in the head and face, especially at night. He complains of pain in the back of the head and neck, indigestion, sour stomach, gas, with belching, and gas in the lower bowel. He is irritable, mentally depressed and lives under high tension. He has increased blood non-protein nitrogen; blood pressure 220 systolic. The Wassermann reaction is negative. He has been refused an insurance policy on his life. He has had all the usual forms of hospital and office treatment, and has been treated at sanatoriums and springs without much improvement in his general condition. He has received foreign protein injections, which did not effect any marked improvement that could be determined by either the patient or his physicians.

He received ten injections of the heart antitoxin serum, which were followed by progressive improvement in symptoms and clinical findings.

Results.—Six months after treatment the blood pressure remained constantly below 150 mm., and there was a complete change in his general appearance. The symptoms had entirely disappeared. After a year an insurance company has issued a large policy on his life.

CASE XI.—Chronic Intestinal Stasis with Arteriosclerosis.—W. M., sixty-nine years of age. This patient showed high blood pressure, cramping of the calves of the legs, much swelling and lameness of the neck and back, contracted liver outline and kidney insufficiency. Inasmuch as his mother died at the age of thirty-seven years from a paralytic stroke, a study was made of his general metabolism. The blood showed increased non-protein nitrogen. He had had some prostatic trouble, for which he had been previously treated. He had been treated in England for general arteriosclerosis by the usual methods. He had also been treated by the injection of bacterial filtrates, vaccines and foreign proteins. The high blood pressure con-

tinued and the nitrogen content of the blood increased. The pains in the neck, back and joints and the cramping persisted, preventing him from using his legs.

He was given a course of heart antitoxin serum injections covering a period of three months, receiving nine injections in all.

Result.—The blood pressure returned to normal. All the unpleasant symptoms disappeared and there was an increase in strength. There was a reduction of the blood nitrogen. After eighteen months the patient was again examined and found to have no return of his former condition. He now enjoys good health and is very active in his business.

CASE XII.—*Multiple Arthritis.*—Miss E. B. L., forty-four years of age. This patient complains of multiple arthritis, which has been confirmed by X-ray examination. She has received injections of vaccines (streptococci), (foreign proteins, etc.), for three or four weeks without experiencing relief. After being treated by a number of regular physicians she still had her joint troubles, which, as she says, "Come and go." Doctor A., an irregular, had given her massage of the spine, and she had received chiropractic, osteopathic and homeopathic treatment. She improved each time after the treatment, but then became worse. She was, at the time the writer saw her, under the care of an osteopath. She states that nearly every joint in her body is affected. She gives a history of having had indigestion for many years, but is now more or less free from this trouble. She has never been constipated and sleeps well. She suffers from general depression, as well as from the severe pain. She had been unable to stand on her feet and has had to quit work. She has been given "foreign protein" injections by a regular physician. These have been followed by reaction, chills, etc., every other day. Thus far, however, there has been no improvement, though at times she states that she experiences a little relief from the massage. (How far these treatments might be instrumental in breaking down tissue and producing autogenous homo-tissue vaccines is a question that must be considered.)

The case had been diagnosed as one of osteoarthritis by a number of physicians who made X-ray examinations.

After having received a course of injections of heart antitoxin serum during a period of a month, she states that she begins to feel her strength returning and has an increased ability to perform work. Her

feet still pain, which is probably due to fallen arches. She has been wearing arch supports, and these latter were discarded. She is now able to work at her sculpture as formerly. The pain in the neck has practically disappeared. The arms and shoulders show great improvement. Gradually all her symptoms disappeared, and she says, "I have a very good report to make and feel very happy over it." At this time she was given another injection of 0.5 c.c. of the antitoxin serum in 1 c.c. of water. A week later she was given an injection of 0.8 c.c. of the antitoxin in 1 c.c. of water. She states that none of the other treatments she has taken have given her the relief that she has experienced from this antitoxin. She is free from the arthritis and her general health and vigor are greatly improved. She is able to attend to her exacting artistic work with ease and comfort, and has no return of her former condition.

CASE XIII.—*Multiple Neuritis*.—Miss A. B., fifty years of age, a miniature painter.

The patient's personal history as given by her is as follows: "I have been painting for twenty-five years and have neuritis in my arms. I have had it for ten years in a mild form. More recently I have been able to work only a few hours, and lately only one-half hour, when the pain would become too severe to permit of further work. Last year I attempted to do a little more and had an acute attack. Last March the pain became so severe that the only way I could bear it was by lying down. It continued in this state for three or four weeks, and then began to ease up. In the middle of the summer I again began to suffer with the left arm, and later with the right. After several weeks of agony I went to the Sprague Institute and received eight bakings. The pain was then almost entirely gone, though I could never use my arms much. My neck and shoulders are also very stiff. The pain and stiffness had again returned. I was also treated by a specialist in Boston for two years, receiving dietetic treatment. I improved somewhat. I have had all my teeth X-rayed and my nose and throat treated. General exercise and leaving out meat during the past two years has improved my general condition somewhat. Whenever I attempt to work, however, the pain and stiffness return."

This patient received from seven to ten injections of foreign pro-

tein without any benefit. She had headaches after menstruation, and sometimes her other trouble is worse at this time.

This patient received six injections of the antitoxin over a period of two months, when all the symptoms disappeared.

Six months later she was examined and found to be completely restored to health and able to attend to her work without pain or distress.

CASE XIV.—*Multiple Neuritis Following Influenza.*—Mrs. J. C. T., forty-eight years of age. This patient was suffering from multiple neuritis following influenza, and stated that she had had urticaria for years. She is troubled with aphasia. Her hair is falling out, menstruation is irregular, accompanied with headaches. This condition had existed for some time and treatment with internal secretions has not afforded relief. The neuritis affected the arms, neck, back and sciatic nerves, causing great suffering and inability to work.

She was given six injections of heart antitoxin serum over a period of two months. The neuritis began to clear up after the second injection, and improvement has been progressive and rapid until the patient is now cured.

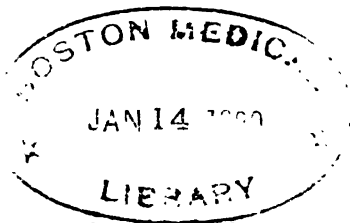
As has been observed in other conditions, the serum H seems to correct endocrine glandular symptoms. After the course of antitoxin was completed and the cure effected, thyroid extract was prescribed, to be used at stated intervals. The neuritis has completely disappeared, as well as the other chronic symptoms above described.

The grouping of these various pathological conditions will sufficiently indicate the rôle that cell necrosis plays in the etiology of disease. It also emphasizes that the rational procedure in correcting a large class of these metabolic diseases is to immunize against the products of cell necrosis, and thus enable the organism to resume its physiological activity. Considerable clinical work is being carried on in various hospitals and clinics which corroborates in every particular my own results. It is all based on carefully conducted experimental research covering a period of twenty-five years. The more recent practical application of this work is the direct result of first producing acute and chronic pathological conditions in animals and then controlling such conditions with specific antibodies.

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SOME PULMONARY CONDITIONS SUGGESTING TUBERCULOSIS AND WHICH MAY BE MISTAKEN FOR SAME

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ALTHOUGH, as we all know, pulmonary tuberculosis is by far the most frequent chronic affection of the lungs, yet the physician must always bear in mind that he may, at any time, meet with conditions suggesting both in symptoms and physical signs tuberculosis, but which have an entirely different etiology, as the following cases will illustrate:

I. A CASE OF PULMONARY ACTINOMYCOSIS

W. J. H., Aetat. forty-four, had previously always been well and actively engaged in his business of an expressman. Without apparent known cause he developed severe pain in the right middle axillary region, extending in front over the region of the middle lobe, accompanied with acute symptoms and an irregular temperature, going as high as 104° and 105° F. on several occasions. The diagnosis of pleurisy, and later of pneumonia, was made by the attending physician. I saw him for the first time some six or seven weeks after the beginning of his illness. He was in a local cottage hospital, with a daily temperature of 98° to 101° and a pulse of 104. His general appearance was fairly good and his principal symptom was a cough spasmodic in character, with copious muco-purulent expectoration, sometimes accompanied with a little blood. There was also some dyspnoea. On physical examination over the upper right front there was diminished respiration, and over the middle lobe there was dullness, bronchial respiration, bronchophony and abundant medium-sized moist râles. Over an area in the middle axillary region there was amphoric breathing. A diagnosis of abscess of the lung, of the right middle lobe, was made and an exploratory puncture advised. This was done later, I learned, without result.

The man gradually improved in general condition, his fever sub-

sided, and he was able to do a little work. The cough decreased but did not disappear. The examination of the sputum at about the time of my first visit was negative to tubercle bacilli, but contained many elastic fibres, desquamated epithelial cells and a large amount of pus. Cultures from the sputum showed a growth, consisting principally of streptococci and pneumococci. As time went on the pain in the right side gradually grew more severe, so much so that he was unable to sleep. Some months later he went to Florida for the winter, where he developed a series of abscesses in the right axillary and cervical regions. The local physician who attended him made the diagnosis of either tuberculous glands or Hodgkin's disease. I saw him on his return from Florida and he was in a truly pitiable condition. He was suffering from a series of suppurating abscesses, had great pain and was coughing spasmodically, with bloody muco-purulent expectoration.

On physical examination there was more or less dulness over the whole of the right chest, which was absolute below the angle of the scapula, bronchophony and many moist râles. He was advised to enter the hospital for the treatment of his abscesses, which he did, and he died about a month later.

While in the hospital the diagnosis of actinomycosis was made for the first time, the fungus being found in the discharge from the abscess. If the exploratory puncture made in the early stage of the disease had been successful, the organism might have been detected then and by surgical interference there might have been a possibility, though not a probability, of arresting the disease.

It was learned, in investigating some possible source of the infection, that the man had been in the habit of chewing straw and pieces of grass while his horse was being harnessed.

II. NEW GROWTHS IN THE LUNGS

CASE I.—R. M., Aetat. seventy, was a well-preserved old man, who had always been fairly well and active in business. He had never had any serious illness. Six or seven weeks previous to my first visit he began to have a little hacking cough, which had progressively increased so that he was unable to sleep at night and was losing both weight and strength. There was only slight mucoid expectoration, which was once or twice blood-streaked. There was some dyspnoea. He obtained the most relief from his harassing cough by lying on his

back or right side. There was no rise of temperature; the pulse was 92, and the blood-pressure 124 systolic and 75 diastolic.

On examination his appearance was that of a well-nourished man, in a fair general condition. There was marked dulness over the left front down to about the fourth rib, with broncho-vesicular respiration and increased voice. Otherwise the examination of the lungs was negative. There was no other evidence of disease. The X-ray showed a dense mass filling the upper half of the left chest, sharply outlined. The examination of the sputum was negative to tubercle bacilli, but showed numerous pus cells. The blood count showed 17,200 white, and the blood smear was negative, with the exception of the leucocytosis. The Wassermann test was negative.

Subsequent X-ray studies disclosed no additional evidence other than a dense homogeneous, sharply outlined mass in the upper chest.

He grew progressively weaker, developed some temperature; the cough persisted, and in a month or two he died.

Aneurysm was considered a possibility, but there was no pulsation to be seen or other evidence. Incapsulated fluid was also considered and an exploratory puncture might have been tried, but the clinical history and physical signs did not lend themselves to this diagnosis. Tuberculosis was ruled out from the absence of tubercle bacilli and other symptoms. Unfortunately no autopsy was obtained.

X-ray treatment was given, but without results.

The diagnosis of a primary new growth in the lungs would appear, from all the evidence, to be well established. The one outstanding symptom in this case was the constant and harassing cough, undoubtedly caused by pressure.

CASE II.—L. H. S., Aetat. forty-five, contractor and engineer. Married.

Family history negative as to tuberculosis. His father died at the age of forty-five, cause unknown, and his mother at the age of sixty, of "tumor." He had always been well and strong and a hard-working business man. So far as known his habits were good. Four weeks previous to his first visit he began to cough and complain of severe pain in his right chest located about the middle lobe. There was slight expectoration, which at times was bloody. He had lost a little weight, had some dyspnoea on exertion, and did not feel as strong as usual, although he was attending to his business.

On physical examination there was slight dulness and diminished respiration over the upper right chest, and a very few fine moist râles in the right supraspinous fossa. Otherwise the lungs were negative. There was no evidence of disease elsewhere. The sputum showed no tubercle bacilli and the predominating pathological organisms were those of the streptococcus-pneumococcus group. The X-ray showed a thick shadow over the upper half of the right chest and much-enlarged mediastinal glands. The Wassermann test, several times repeated, was negative.

About two weeks later he was seen again, and meantime his cough and expectoration had increased, and on one occasion he spit up a little blood. While on a business trip to Washington he said that for two or three days he had fever and his symptoms were aggravated. He also bitterly complained of pain in the right upper chest, which was relieved by strapping. The physical examination was about as before.

Ten days later he was seen at his home, where he was confined to bed, on account of a rise of temperature, sometimes as high as 103°. The cough had increased with a tenacious mucoid sputum; the pain, however, had disappeared, the cause of which will appear later. On examination the resonance in the right upper chest was diminished, as likewise the respiration, and there was a diffuse bronchitis.

About a month later he was again seen. Meantime he had been having an irregular temperature and had lost flesh and strength. There was no pain and the cough was less severe. On examination there was very marked dulness over the upper portion of the right chest, particularly over the middle lobe, and absence of respiration. Otherwise the lungs were negative except for a few bronchitic râles. The blood count showed a leucocytosis of 25,000. An exploratory puncture in the upper axilla was made and a few drops of thick bloody pus were obtained. Two days later a portion of rib was excised and the pleura opened and about a pint of thick pus was evacuated and a drainage tube inserted. The temperature dropped to normal and the general condition improved. It was believed that the cause of the trouble had been found and remedied, and hope was high that a speedy recovery would ensue.

Such, however, was not the case, for about three weeks later the patient developed cedema of the arms, hands and neck. Of course,

this was recognized as the result of pressure upon the vessels, and the question was as to the cause of this pressure. Was it a tumor or a deep pocket of pus which had not been reached and drained? The X-ray taken at the time showed a dense mass in the upper right chest extending over towards the left. The physical examination showed absolute dulness and absence of respiration over this area.

An enlargement of the original opening was decided upon with a thorough exploration of the cavity, and also an exploratory puncture in front, with the object of discovering and evacuating a deep interlobar abscess, if it existed. This was done, but no deep abscess was discovered. Quite profuse hemorrhage occurred, so much so that the cavity had to be packed.

Three weeks later he was seen again, when the change for the worse was very striking. There was very considerable œdema of the face, arms, hands and chest, and a chain of enlarged glands along the right side of the neck extending down to the clavicle. The veins on the right chest were prominent. The man was much emaciated and was almost aphonic. At short intervals he had severe and exhausting paroxysms of coughing. The physical examination of the chest showed extreme dulness on the right, extending over to the left, and absence of respiration. The blood smear showed 70 per cent. polynuclear cells and 30 per cent. lymphocytes; there was no apparent leucocytosis. The sputum was bloody and purulent. Microscopic examination showed pus cells, but no tubercle bacilli. No actinomyces granules were found.

Three weeks later the general condition had not materially changed except that there was quite an abundant discharge from the wound and some diminution of the œdema. There was also marked somnolence. A few weeks later he died, and, as with so many cases in private practice, no autopsy was obtainable.

In arriving at a probable diagnosis there were two possibilities: (a) a new growth; (b) an interlobar abscess or encysted empyema.

Considering the history, the symptoms, especially the pressure symptoms, the significant chain of glands in the right cervical regions, and the general course of the disease, the existence of a new growth, probably carcinoma, seemed the only diagnosis which could account for all these symptoms and the progress of the disease. The interlobar abscess or empyema, which we hoped was the main condition,

can be ascribed to a secondary infection of the pleura. Moreover, an encysted empyema or interlobar abscess would not in all probability give rise to pressure symptoms such as were observed in this case, or to the glandular enlargement. As there was no evidence of malignancy in any other part of the body, this case must be considered as a primary new growth in the lung.

The following case is of interest in connection with the two previous ones, especially the second. The predominant symptom and the X-ray picture were very suggestive of the same condition which existed in the two other cases, but the result was very different. It also teaches the lesson that one should not put too much confidence in the X-ray alone in making his diagnosis.

CASE III.—N. L., Aetat. thirty, a well-nourished man weighing about 200 pounds, gave the history of having had various hemorrhages for the last six years, most of them slight, and an intermittent cough. He had been examined many times by many physicians, sometimes with a negative and sometimes with a positive diagnosis of pulmonary tuberculosis. Five years ago he was advised to enter a sanatorium. The sputum had never been positive. All this time his general condition was good, and for the most part of the time he was attending to his business. For quite six months while he was under observation he had an almost incessant cough both day and night, and no treatment or change of climate had more than a very ephemeral influence upon it. From time to time he had a little temperature, which never lasted long. The sputum was frequently streaked with blood and was negative, as it always had been, to tubercle bacilli. His general condition remained good; he would alternately lose and gain a few pounds. The physical examination was generally negative, although on one occasion a very few râles were discovered in the right supraspinous fossa after the cough, and on another occasion a few bronchitic râles in the right back. The X-ray showed a marked shading about 5 or 6 metres square over the middle lobe of the right lung.

As will be observed, the X-ray picture was quite similar to that of the previous cases, only it was not so dense. With this evidence, together with the incessant and intractable cough, the varying temperature and the slight but quite frequent hemorrhages, several diagnoses were suggested: a new growth, a tuberculous process, a bronchiectasis, or a simple pleuritic thickening.

At the end of about six months the cough entirely disappeared, with all symptoms, and the man appeared and felt as well as ever, and remains so to this day, and recently reported himself as well and attending to his business.

What did the X-ray picture indicate, which, as I have said above, closely resembled that of the previous cases?

One other possibility should have been considered, namely: syphilis, and a Wassermann test should have been made.

III. BRONCHIECTASIS AND FETID BRONCHITIS

CASE I.—Mrs. L. B., Aetat. forty, was referred to me by her surgeon as a case of pulmonary tuberculosis, with a cavity at the right apex. As will be shown later, his physical examination was correct, but his diagnosis was wrong. She was a fairly well-nourished woman, weighing about 190 pounds, and appeared to be in average good health. She gave the history of having suffered for the past seventeen years from a paroxysmal cough accompanied by an ill-smelling expectoration. The onset of this seventeen-year cough she referred to a date shortly after the birth of a child. She described the cough as intermittent and always accompanied by quite profuse fetid expectoration. Whenever she stooped down or leaned over, there was a gush of sputum which was sometimes blood-streaked. There was more or less dyspnoea on exertion. She was unable to lie on her right side.

The physical examination showed marked dulness throughout the left lung, with bronchial and broncho-vesicular respiration, and a variety of moist râles. At the upper portion of the left lung there was evidence of a cavity. The sputum, several times examined, was negative. The whole of the left lung was involved, and from the physical signs alone one would make the diagnosis of advanced pulmonary tuberculosis, which was the diagnosis of the surgeon who referred the case. It was, however, one of bronchiectasis of long standing, with extensive disorganization of the lung, and the diagnosis is established by the absence of tubercle bacilli in the sputum, the paroxysmal cough and profuse fetid expectoration and the general well-being of the patient. If pulmonary tuberculosis had existed for seventeen years and the destructive process had become so extensive, as in this case, the woman would not have exhibited the appearance

of ordinary good health which she did. Indeed, she would not have been alive.

The case teaches that when on repeated examinations tubercle bacilli are absent, one must look for some other condition to account for the physical findings than tuberculosis.

CASE II.—F. K., Aetat. forty-two, rag merchant.

Family history: his father died of some pulmonary disease, and his mother of cancer.

He had always been well, with the exception of pneumonia twenty-four years ago and again four years ago. Two months previous to his visit to me he had some acute pulmonary affection, indefinite in character, so far as any accurate description could be obtained from him. It was evidently not very severe. Since that time he had been suffering from paroxysms of coughing both night and day, with profuse and very malodorous sputum. From time to time he had had slight hemorrhages. He was unable to lie on his right side. He was fairly well nourished, had lost but little, if any, flesh, and had a good appetite, although the intolerable odor and taste of the sputum sometimes interfered with his taking of food.

Upon physical examination, below the angle of the left scapula there was marked dulness and distant respiration, with no râles. Otherwise the lungs were negative. In the sputum there were no tubercle bacilli, but a large amount of pus and a few organisms of the strepto-pneumococcic type. On standing the sputum showed the three characteristic layers of fetid bronchitis. The X-ray showed only an increase in the size of the glands around the left root extending somewhat into the lung tissue and downward toward the left diaphragm, which was higher on the left side than normal. There was not sufficient evidence that there was any fluid, free or incapsulated. There was the possibility of a thickened pleura at the left base. The fingers were slightly clubbed. From the result of the physical examination, namely, dulness and distant respiration, an exploratory puncture was made over the dull portion of the lung at the left base, but without results.

On obtaining further history it was learned that four years ago, after the pneumonia, he had symptoms similar to those at the present time, namely, a paroxysmal cough with fetid expectoration. The diagnosis of bronchiectasis is made from the absence of tubercle bacilli

in the sputum, the location of the physical signs at the base, the paroxysmal cough and the fetid sputum, and the fact that the disease had made so little impression upon the man's general health. One might ask: Why not a simple fetid bronchitis? It may have been, but there could hardly have been so profuse a sputum and a history of a previous attack four years previously, together with the physical signs, without some bronchial dilatation. From the physical signs one would have expected a different X-ray picture.

The above cases, actinomycosis, new growths, and bronchiectasis, are of interest not only in themselves, but from the fact that anyone of them might be mistaken for tuberculosis, as indeed two of them were. While, of course, pulmonary tuberculosis is far more frequent than any other disease of the lungs, yet other conditions sometimes occur, and one must always bear in mind the possibility of their occurrence when on repeated examinations tubercle bacilli are not found in the sputum.

These cases also teach us the value of the X-ray in diagnosis, and at the same time that it is only one factor in the complex of the symptoms and signs, and should not solely be relied upon.

Finally, one may add that the possibility of syphilis should always be kept in mind.

CASES ILLUSTRATING DIFFERENT KINDS OF BONE SURGERY

By WILLIAM HESSERT, M.D., F.A.C.S.

Professor of Surgery, Chicago Polyclinic; Attending Surgeon, St. Joseph's, Alexian Brothers, and Polyclinic Hospitals, etc., Chicago, Ill.

I SHALL exhibit before you this evening a number of cases which illustrate various types of fractures and operations for their cure. There is nothing unusual about the cases or the success of treatment. They are picked more or less at random, but yet with the intention that each case should bring out some particular point or teach some useful lesson.

CASE I.—*Fracture of the humerus three years ago; musculo-spiral paralysis; nerve suture; complete functional recovery in seven months.*

The first patient is a man of fifty-nine years, who was injured three years ago. He fell down stairs and sustained a spiral fracture of the right humerus at its middle. He had an immediate musculo-spiral paralysis—a complete wrist-drop. Musculo-spiral paralysis is one of the important nerve complications of fracture of the humerus shaft. It may be primary or secondary. Primary paralysis, in the vast majority of cases, is due to a more or less complete division of the nerve. The symptoms are immediately apparent after the injury. I cannot exaggerate the importance to the surgeon of determining the presence or absence of wrist-drop in fractures of this kind, and to do so before any reduction is attempted or dressing applied. There was a case in court not long since in which the plaintiff claimed the wrist-drop was the result of faulty dressing by the doctor. The defendant did not himself know, nor could any witnesses be produced who could testify as to whether the paralysis was present or not before treatment was begun. The verdict was for the plaintiff.

Secondary paralysis makes its appearance one or more weeks after the injury. The wrist-drop may be just as pronounced as in the primary type. It may be due to contusion only, or to continuous pressure by a bony edge or loose fragment, scar tissue or callus or

hemorrhage. Both types are exactly similar clinically when fully developed, and the only means of differentiation is by means of the history or personal observation. This is of vast importance, for the primary paralysis generally does not recover spontaneously, and the secondary type usually does make a recovery without operation.

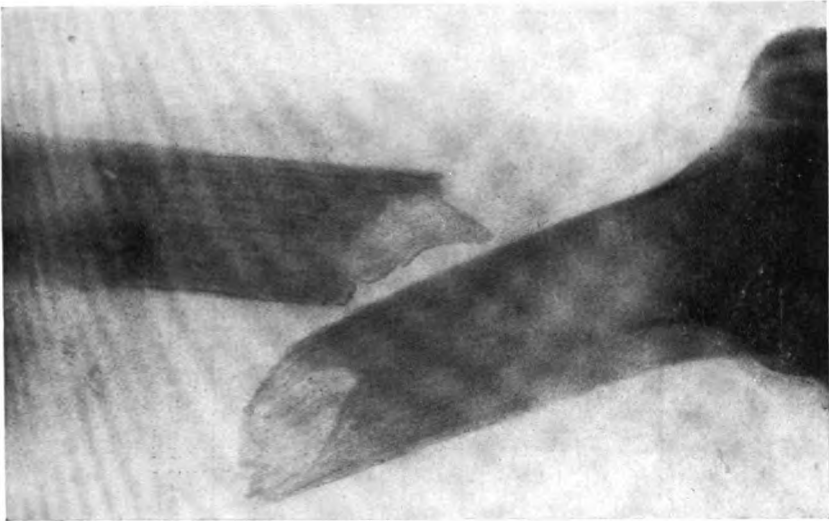
It is my practice in cases of primary paralysis to advise early operation, and in my experience, this has thus far proven justifiable, for I have invariably found the nerve either completely or almost severed. In the secondary type I advise waiting four to six months, at which time I would advise cutting down on the nerve if there has been no improvement.

This man was operated one week after his injury, not primarily for the reduction of his fracture, but to investigate the condition of the musculo-spiral nerve. This was found completely severed and was sutured with silk. The bony fragments were held in place by means of a Parham-Martin band. He made an uneventful operative recovery, and the interesting feature to-day is the demonstration of his complete recovery from the paralysis. There was practically no sign of return of function for about three months. After that time motion returned slowly, assisted by massage and electric treatment, until after seven months, motion of the hand was normal. Sensation was slightly involved for some time longer. During the period of regeneration of the nerve the nutrition of the muscle must be sustained not only by massage, but above all things, you should maintain the hand supported. Do not let the hand hang down, but carry it on a suitable splint. Failure to do this may result in fibrous changes taking place in the muscles of the entire forearm, leading to a condition not unlike Volkman's paralysis, with the result that the regenerated nerve is unable to activate the degenerated muscles.

CASE II.—Simple transverse fracture of the femur shaft; Lane plate; good union; plate breaks before cast is removed; removal of plate.

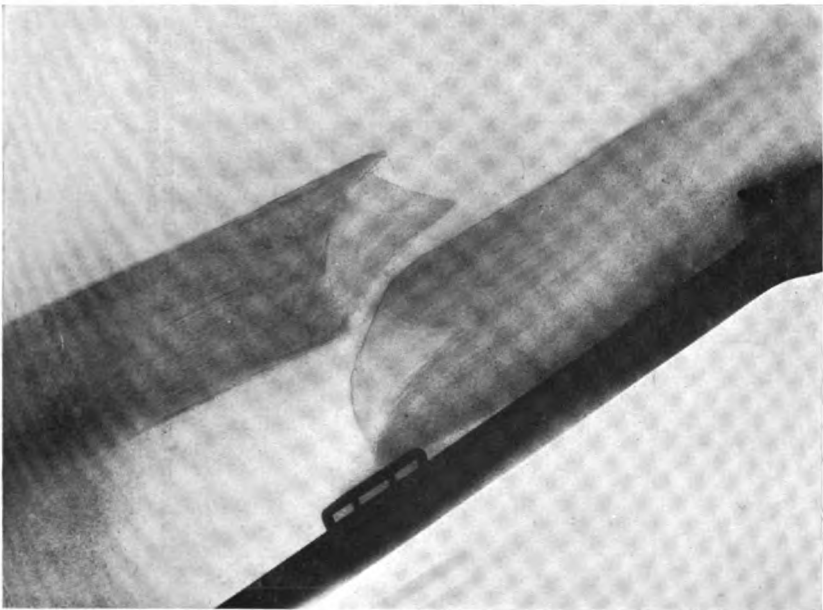
This is a young man of thirty years, who was injured six months ago while chopping down a tree. He was brought to my service at the Alexian Brothers Hospital suffering with a simple fracture of the femur (Fig. 1); it was a transverse fracture in the lower fourth. It was too high up to be called supracondylar. My experience with transverse fractures of the femur is that the great majority require

FIG. 1.



Oblique fracture of femur with typical backward displacement of lower fragment due to contracture of calf muscles.

FIG. 2.



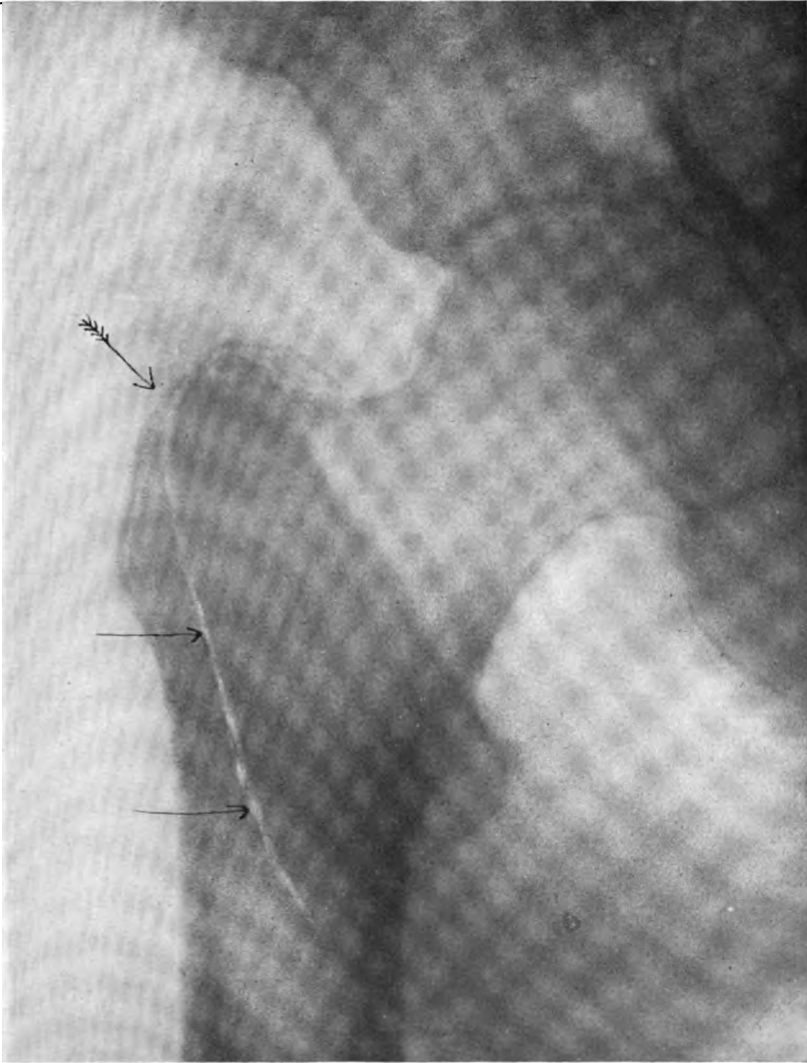
Longitudinal shortening has been corrected by extension, but lateral displacement persists. This was shown to be due to interposition of muscle and fascia. The leg is in the railroad splint, and traction was maintained while radiogram was taken.

FIG. 3.



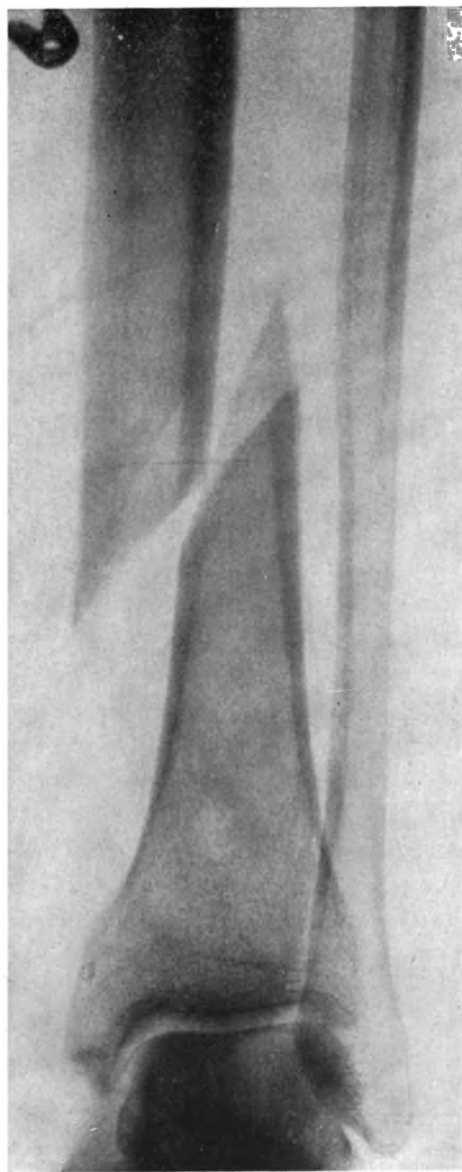
The broken Lane plate is shown. The bones are in good position. Röntgenogram was taken about six weeks after operation. There is an abundant callus, and union was already quite firm.

FIG. 4.



A case of incomplete fracture. The fissure is shown beginning at the upper border of the greater trochanter and running obliquely downwards, finally becoming indistinguishable.

FIG. 5.



Typical right-handed spiral fracture of the tibia; such a one as is produced by torsional violence. There is lateral displacement with about three-quarters of an inch shortening. The sharp ends of bone in this class of fractures are almost invariably speared into the adjacent muscles, making accurate reduction impossible.

open reduction. Oblique fractures are capable of reduction satisfactorily by some form of traction apparatus, but in transverse fractures the overriding is almost impossible to correct. I have demonstrated at operation the futility of traction in these cases. With the patient fixed in the Hawley table, and the fracture exposed by incision, no amount of traction alone would ever reduce the fragments. It was always necessary to guide the fragments with bone forceps, after disentangling the interposed muscles. Remember also that traction is in a straight line, and the femur is normally bent like a bow. To reduce a transverse fracture at operation, the best way is not to employ traction at first, but to grasp the fragments with forceps, approximate the ends while the fragments are in a V position and then force them back. Then only would I apply traction enough to keep the muscles tense.

According to my experience, if by any form of traction or manipulation you have secured end-to-end apposition of a transverse fracture of the femur, it is a matter of pure luck, and I do not believe you could do it again in a dozen trials. The fluoroscope is a great aid in this regard, but not generally available.

In the case of the patient before you, I gave a general anæsthetic, and after attempting to lock the fragments in an end-to-end position, I placed him in the railroad splint, which I demonstrated here some months ago. Twenty pounds of weight were applied. This railroad splint is a device which works in principle like a double-inclined plane. The radiogram taken from the side shows that the traction has indeed reduced the longitudinal displacement, but not the lateral.

I was unable to correct this, and it was shown later that there was interposed muscle which kept the fragments apart (Fig. 2). An open reduction was done—the interposed muscles were pushed away, the fragments somewhat everted out of the wound and brought into accurate apposition. I doubted that the fragments would remain reduced, even in a cast, so a Lane plate was screwed in place. I felt so sure that everything was in good shape that I ordered no radiogram for about six weeks, about the time the first cast was removed. To my surprise, the Lane plate was broken (Fig. 3). The bones were in perfect position and union was firm, as attested by the large callus. How and when was the plate broken? How was it possible for the plate to be broken without disturbing the fragments? The body cast

was applied after the operation before letting up on the traction, and I make it a practice of doing this myself to avoid any possible accident before the plaster hardens. I am inclined to believe that the patient, who was restless and violent when coming out of the anæsthetic, probably broke the plate at this time, without (fortunately) disturbing the fragments. Moral: Do not fail to take an X-ray picture as soon after operation as possible to control the result.

The plate being broken, there was nothing gained by leaving it *in situ*, so the pieces were removed under local anæsthesia. The man has made a perfect recovery. Motion of the knee is perfectly free, and he is able to mount a chair with the injured leg; in fact, he does everything now that he ever did before. As to Lane plates breaking, I have seen that happen before. This occurs especially in femur cases, in such cases where there has been a sinus. In such cases osteogenesis has been imperfect even after a lapse of two months. The patient makes possibly his first attempt to walk, and the plate breaks. In such a case there has been little if any union by callus, due partly to the low grade chronic infection and partly to the fact that all cases plated require a longer period for the formation of callus. The absolutely rigid immobilization by the plates eliminates the small amount of motion about the fragments which is indispensably demanded by nature to maintain the normal amount of hyperæmia which is conducive to normal callus formation. In cases plated, I always empirically add one-third to the normal time required for union.

CASE III.—*Intertrochanteric fracture of the femur in a man of sixty-five years; incomplete, with no displacement.*

This old man of sixty-five years was injured fourteen weeks ago when he fell on the sidewalk and struck his right hip. He was brought to the hospital unable to walk and suffering great pain in the right hip. He was unable to lift his leg, but there was neither inversion nor eversion of the foot. The region of the hip was swollen and tender; there was no shortening of the leg. Careful passive motion of the hip was free and revealed no crepitus. The trochanter was in normal relation to Nélaton's line. Examination of the pelvis showed no sign of fracture. Dislocation of hip and impacted fracture of the neck of the femur seemed out of the question. The injury had the appearance of an ordinary contusion. The X-ray revealed an interesting condition. There was a fissure starting from

the upper border of the great trochanter and running obliquely downward for a distance of four inches, where it became indistinguishable (Fig. 4). In other words, it was an incomplete fracture. The force was spent before the fracture became complete. The clinical symptoms and findings were thus clearly explained. The treatment was merely "rest in bed for two months." The patient was allowed to sit up in bed and the leg was steadied with sand bags. He has made a complete recovery, and, as you see, he walks without a cane and has free use of his limb. Before the era of the X-ray this injury would have been diagnosed "contusion," and this case brings home to us the importance of always taking a radiogram as a routine procedure.

CASE IV.—Spiral fracture of the tibia; unsuccessful attempt at reduction; application of Parham-Martin band.

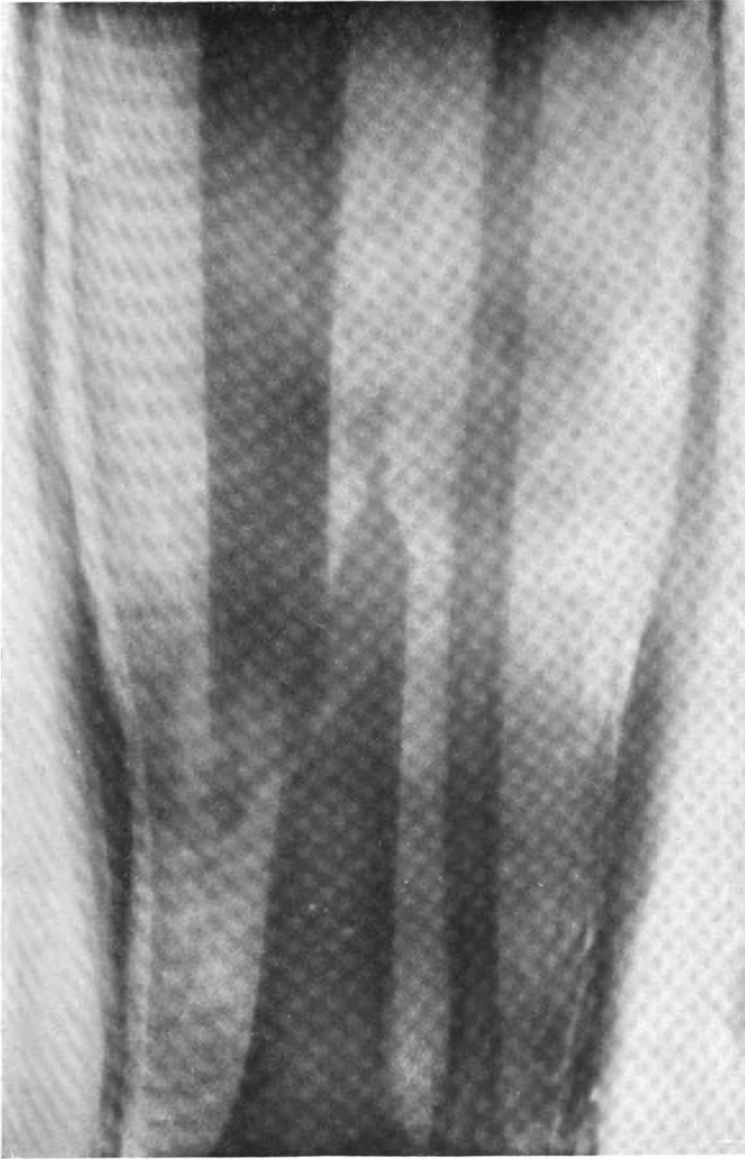
This patient, a man thirty-five years of age, was injured two months ago in a street mêlée. He was brought to the hospital with a simple fracture of the left leg, which the X-ray revealed as being a spiral fracture of the tibia somewhat below the junction of the lower and middle thirds (Fig. 5). There is nothing extraordinary about this case or the treatment; on the contrary, it is a very typical form of fracture and of common occurrence, and it is for this reason that I am presenting him and detailing the treatment. The temporary dressing in this case was a blanket splint. The purpose of any temporary dressing is to immobilize the fragments, thus relieving pain and preventing further displacement. What is the most important factor in causing displacement of the fragments of long bones? The answer is, muscular traction. The pain and irritation incident to a fracture result in a reflex involuntary muscular spasm. Again, when the points of origin and attachment of a muscle are approximated by a fracture with overriding, the tendency for the muscle is to take up the slackness by contraction. Many types of fracture will not stay reduced on account of muscular traction. The reduction of a fracture would be most simple had we not the muscles to reckon with. What causes the displacement in such classic fractures as fracture of the femur at the junction of the upper and middle thirds or above the condyles—of fractures of the clavicle—of the olecranon and a host of others? It is always a case of muscular pull. Take a spiral fracture like the one involving this tibia. Granting

that the sharp ends are not speared into the muscles, and we are able by traction to reduce the deformity, will it stay reduced? It will not. As soon as extension is released, the ends of the bones will slide by and the deformity recurs, due to the muscles. This situation you will say calls for treatment by extension—extension maintained beyond the power of muscular pull. That is fundamentally true, and the method should be employed where practicable.

I believe that in the matter of temporary splints, as ordinarily applied in our hospitals, not enough attention is paid to maintaining some form of mild traction to prevent muscle spasm. Cases of fracture of the shaft of long bones are too often left in some inefficient temporary dressing for a week or more before the bones are "set." After the lapse of a week, cicatricial contraction, consolidation of blood clot about the fragments and in the tissues, and last but not least, a shrinkage of muscle which cannot be overcome—all of these factors often make a fracture impossible of good reduction. Yes, many such fractures can be easily reduced early and held in good position until the swelling has subsided. I urge the more frequent employment of such standard forms of apparatus as the Thomas traction arm and leg splint. These splints have been modified and improved and make splendid temporary dressings, mainly due to the traction. We have also the Jones humerus traction splint, the Hodgen splint and a host of others. I am still referring to their use as temporary dressings. I believe we should employ all forms of approved conservative treatment for the reduction, and maintenance of reduction, of fractures before deciding on operative reduction. In my opinion, operative reduction should be the last resort, after all other methods have failed. A surgeon experienced in fracture work does not necessarily in every individual case try every method he knows of. He does not have to go through the entire routine every time. In other words, he has learned by long experience whether, in a certain type of fracture, conservative methods will succeed or not. He is likely, after a careful survey of the case, to advise operation right from the beginning.

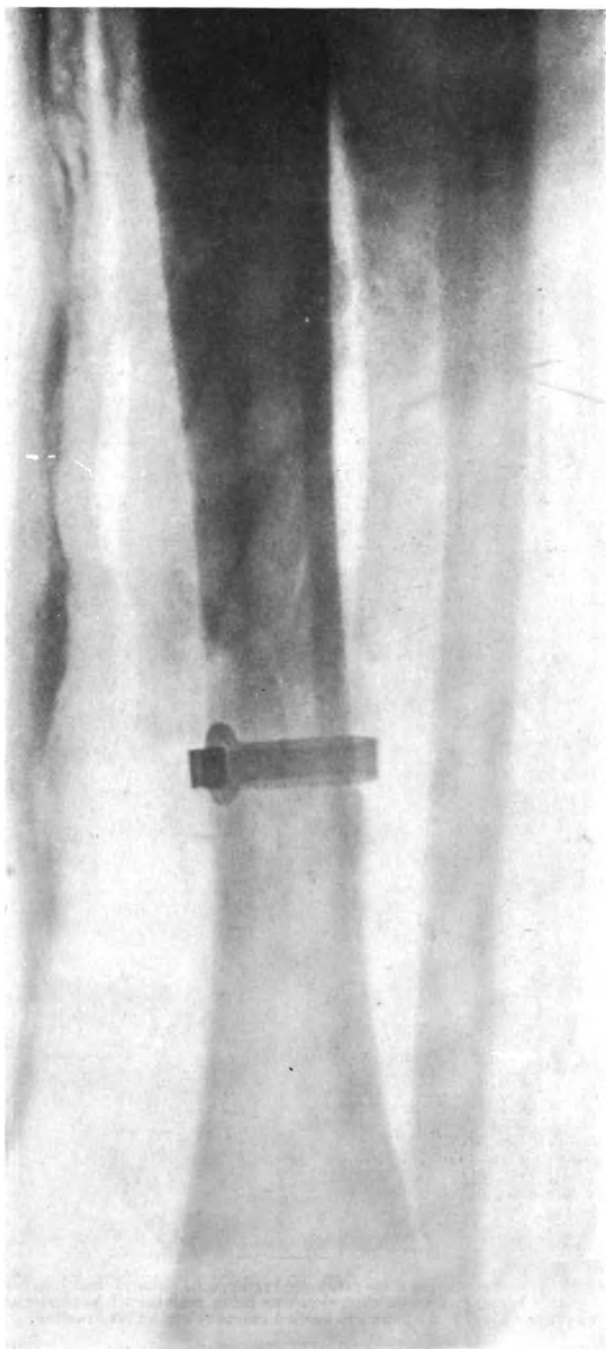
Take a spiral fracture of the tibia such as we have here. My routine treatment consists in the use of a little traction until the swelling has largely subsided—about one week. Then the patient is anesthetized, placed on a Hawley table or other extension appa-

FIG. 6.



This view shows the condition after attempted reduction. The patient had been anesthetized, placed on a Hawley table, and while extension was being maintained, a plaster-of-Paris cast was applied well above the knee, and allowed to harden before releasing the traction.

FIG. 7.



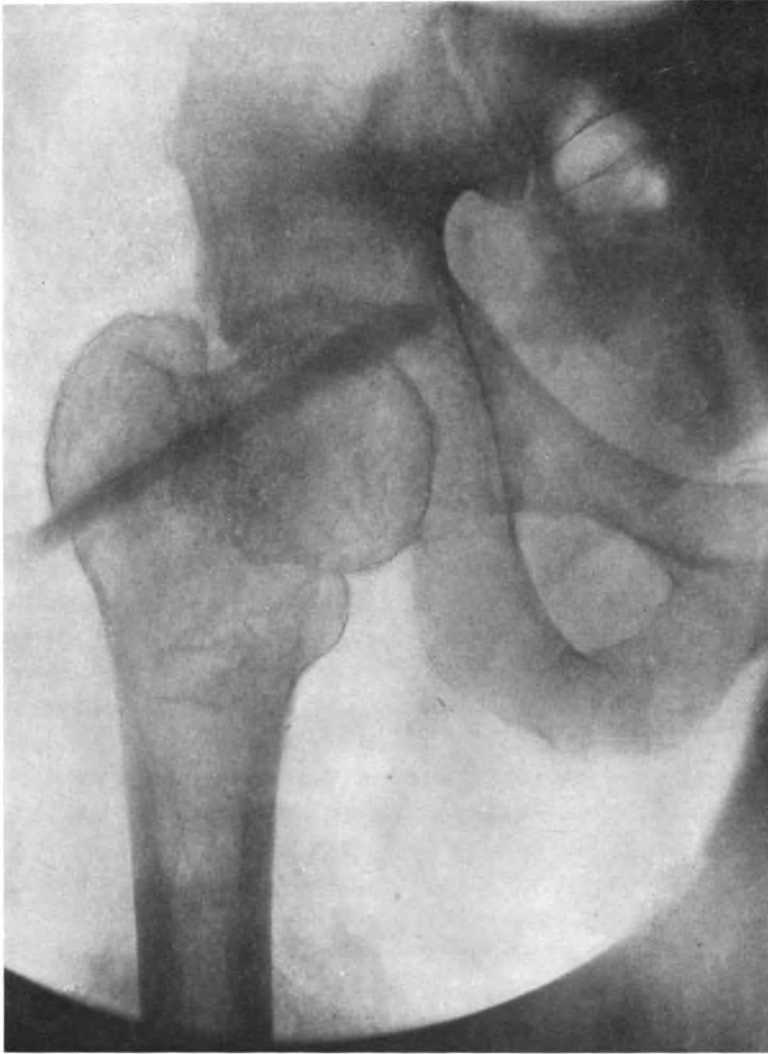
This view of Case 4 shows the result after insertion of a Parham-Martin band. The plaster cast is still in place.

FIG. 8.



Fracture of the neck of the femur of two years' standing with non-union. There has been a considerable absorption of the neck but the matter of non-union can not be determined from the picture alone. Note the over-riding and shortening, and the relation of the trochanters to the neck and compare with the picture taken after operation.

FIG. 9.



Ununited fracture of the femur after operation. By comparing with the previous picture the amount of correction of the deformity can be noted. It might have been better to have placed the peg somewhat lower. But this as well as the projection beyond the head seemed not to have jeopardized the result, which was all that could be wished for.

ratus, and while extension is maintained, a plaster cast is applied, carrying the same well above the knee. The extension is not released until the cast has hardened, after which the foot part is finished. The cast is an illusion in this instance; it does not maintain traction, and when a radiogram is taken next day the bones will be found displaced just as they were before. This is exactly what happened in the case under discussion (Fig. 6). After having failed in my attempt at conservative reduction, I felt justified in advising open reduction. I chose the Parham-Martin band because this device lends itself admirably to the maintaining of apposition in this type of spiral fractures (Fig. 7). The band is not applicable where there is a transverse fracture, but I will say in passing that it is rarely necessary to operate transverse fractures of the tibia. Strangely, just the reverse holds true in case of the femur. As they involve the shaft of the femur, oblique fractures can generally be reduced with a satisfactory result by conservative means, while the transverse type are very difficult to reduce, and in my experience almost invariably are operative cases.

The post-operative history of this case was uneventful. The wound healed by primary intention and the cast was worn for six weeks. From that time onward the patient is allowed to bear more and more weight on the injured limb. The bones of a limb that has been immobilized for six weeks show plainly the result of suspension of function; namely, rarefying osteitis. Disuse results in an absorption of lime salts, a state of affairs that does not promote callus formation. There is such a thing as immobilizing fractures too long—thus arise some of the cases of non-union. When function is resumed; that is to say, weight-bearing function in case of the leg, the bones quickly acquire their former density. This matter of rarefying osteitis is an argument in favor of the ambulatory treatment of fractures. An ideal condition would be "business as usual while undergoing repairs." Most cases of delayed union of the tibia can be cured applying a leg corset and urging weight-bearing.

I will now show you two cases of fracture of the femur treated by operation.

CASE V.—Man thirty-five years old; operated one year ago for an ununited fracture of the neck of the femur of two years' standing. Typical bone peg operation; good functional recovery.

The first patient, age thirty-five years, came into my service at the

Alexian Brothers Hospital about one year ago. He gave a history of having fallen two years previously and injured his right hip. He had a great deal of pain and was obliged to remain in bed for over a month. His physician treated the injury as a contusion, and eventually the patient was able to limp about. He remained greatly disabled, never was able to work and went about laboriously with a cane. He always suffered great pain in the hip.

Examination on admission revealed a normal condition except as to the right leg. Blood and urine normal; Wassermann negative. No history of previous illnesses. He walked with a decided limp on the right side. The muscles of the right leg were somewhat atrophied. The right leg was one inch shorter than the left, and the greater trochanter was elevated above Nélaton's line about the same distance. On making traction on the limb, the shortening could be reduced to one-half inch. There was slight crepitus on rotation. The radiogram (Fig. 8) showed a fracture of the neck of the femur, with considerable absorption of the neck. The clinical examination had shown, however, that not only was there a fracture, but there was non-union. I question the possibility in this case of diagnosing with a certainty the fact of non-union from the radiogram alone. I have many times had no difficulty in establishing the absence of non-union from the radiogram, but it is always advisable to make a careful clinical examination. The physical findings here proved the absence of union. I would advise in all fractures to make as thorough a physical examination as is possible, as though we had not the X-rays to assist us. The radiogram is only a shadow, and can be easily misinterpreted.

There is only one way to cure a case of non-union of the neck of the femur, and that is the insertion of an autogenous bone peg. Formerly we tried prolonged immobilization in this class of cases and failed to secure union. Later we tried holding the fragments with a metallic spike, but found this method about as useless as mere immobilization. Why? Why is there ever non-union in this type of fracture? Is it a matter of inefficient immobilization? No, it is not. It is a matter of disturbance of nutrition of the fragments. The head and neck of the femur receive practically no nourishment by way of the ligamentum teres. The blood supply flows distalward towards the head from the base of the neck. Thus, when a fracture occurs, the loose fragment is almost totally deprived of its blood

supply, and assumes the rôle of an aseptic sequestrum. If the fracture line is close to the head of the bone, then the greater portion of the neck is still attached to the shaft and receives its normal blood supply. In cases of this kind the conditions are most favorable for union of the neck with the loose head. All that is necessary is good apposition firmly retained, as in Whitman's abduction position. There is not much danger of bony absorption from lack of nutrition. In fractures of the base of the neck we have quite another story. The loose fragment consists of the head and the greater part of the neck. It is in this class of cases that absorption of the neck is likely to occur. Absorption from diminished blood supply is incompatible with regeneration and repair. The clinical evidences of absorption and non-union may not become apparent until quite late. Thus the fracture may at first be even impacted. Healing seems to progress and in due time the patient is up and around. After a few months, instead of improving, the condition seems to get worse. There is more pain, more disability and more shortening. Eventually after eight or ten months there is great disability and pain. An X-ray then will show a disappearance of the neck, sometimes of part of the head. There is no sign of union and the trochanter major has slipped way beyond the rim of the acetabulum, resulting in an inch or more shortening.

Now, in such a case, is immobilization going to do any good? Of course not. Is immobilization with a metallic spike going to do any good? Certainly not. The presence of metal about a fracture is bad enough in cases where there is no question as to nutrition. But here it only adds insult to injury, and in my experience these spikes all had to come out eventually and the condition was as bad if not worse than before. I will say nothing of the joints that were nailed where the spike missed the head entirely. Then what is the indication? The indication is to supply that which is deficient in these cases; namely, active proliferating bone. You must make up for the lack of osteogenetic ability of the distal fragment. This is done by joining the fragments with an autogenous bone graft. Just what becomes of this bone graft is a matter of some dispute. I believe that a portion of it is absorbed. Part of it serves as a supporting framework along which blood-vessels bud and elongate, and in which osteoblasts work their way and multiply. I believe, further, that a large number of

the osteogenetic elements of the transplant proliferate in the graft and in its periphery, fusing the graft with the surrounding inert bone, and lending to the latter the active elements necessary to live bone. Once a live bridge of bone is established between the fragments, then the outcome is assured. The reestablishment of function, motion and weight-bearing, in accordance with Wolf's law, will automatically strengthen and augment the seat of fracture until there is full restoration. Clinically, this is manifested by absence of pain and restoration of function.

Now, to return to our patient, we had here a case of fracture of the neck of the femur with non-union of two years' standing. The operation performed was the typical transplant operation. The patient was placed on a Hawley table and extension applied to the legs. An incision was made along the inner edge of the sartorius muscle and the joint exposed. The joint cavity was opened by incising the capsule, bringing the fracture in view. The fractured surfaces were freshened with a curette. The leg was then placed in a position of abduction, and sufficient traction applied to correct the shortening. An incision was then made over the trochanter major. Then with a motor saw a graft was removed from the crest of the tibia. This was about four inches long and one-half inch square. It was purposely denuded of periosteum. I omitted to say that before preparing the graft, an electric drill was employed to drill a hole through the femur into the head. With a burr the tunnel was enlarged sufficiently to receive the graft. Then it became a simple matter to insert the graft and drive it home into the head of the bone (Fig. 9).

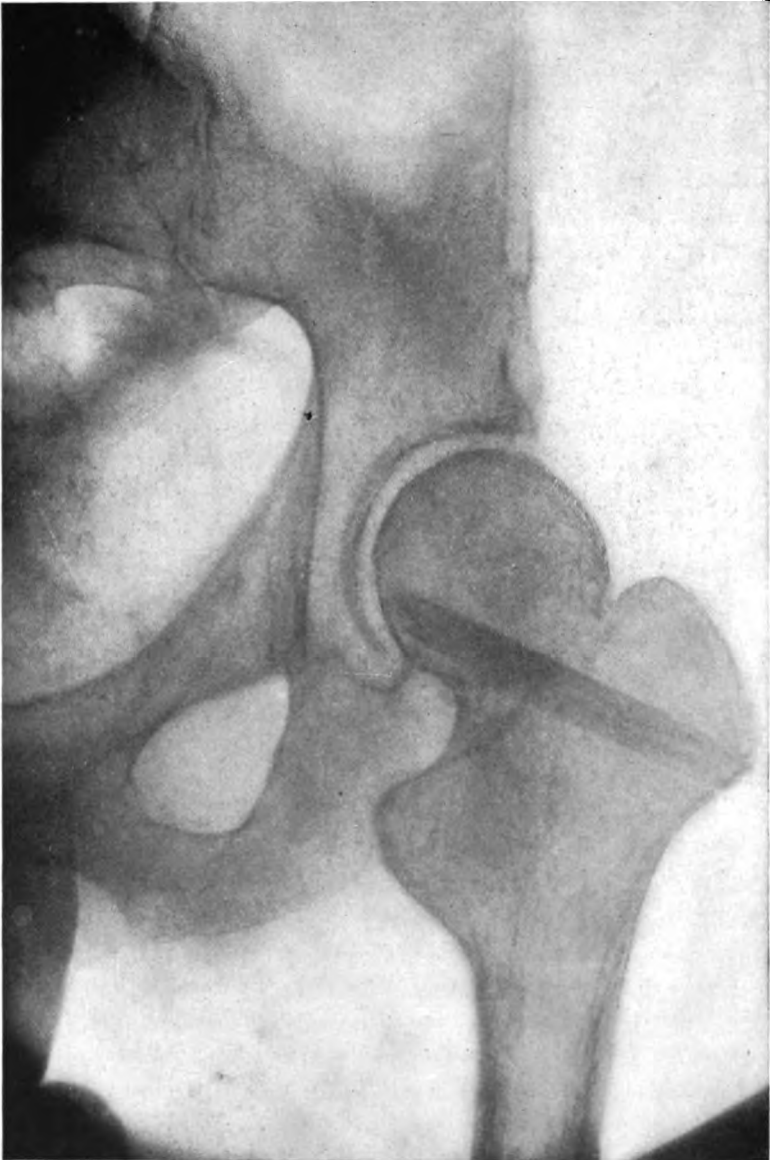
The wounds were sutured and the patient encased in a body cast. Convalescence was uneventful. The cast was worn about three months, during most of which time the patient was up and around on crutches. After four months he began to bear more and more weight on the leg, and in six months he was walking without support. I present him now after the lapse of one year. He has no pain and walks with only a slight limp. He has been back to work several months and is able to be on his feet all day. The radiogram (Fig. 9) is not a recent one, but was taken after the cast was removed. It shows the graft in place, and I will call your attention to the upper end of the peg which projects slightly beyond the head of the bone. I was a bit concerned about this at first, but it seems to have made no

FIG. 10.



Fracture of the neck of the femur of three months' standing. The head of the bone is tilted and the neck has been entirely absorbed. There is no union.

FIG. 11.



Same as Fig. 10, three months after operation. The deformity has been corrected and union has taken place, but there is scarcely any neck to be seen. The parts are in as good a relative position as is possible. The graft still shows plainly, owing to its greater density. After three months the graft shows no signs of absorption. This peg also might well have been placed a little more obliquely upwards. Function was perfect in spite of three-quarters of an inch shortening.

difference with function. Eventually this projecting portion will be absorbed. You see that motion at the hip is quite free in the direction of flexion and rotation. When he walks his foot is not rotated outward, as is so often observed in fractures of the hip. Adduction is rather limited. He is able to walk up stairs normally, but cannot as yet step onto a chair with the injured leg. There is about one-half-inch shortening, which is overcome by an inlay in his shoe.

CASE VI.—*Man forty-one years old; operated one year ago for ununited fracture of the neck of the femur of three months' duration. Bone-graft operation, with perfect result.*

Our next patient is a man of forty-one years, who came to my clinic at St. Joseph's Hospital about one year ago. He gave a history of having fallen three months before and injured his left hip. His injury was not treated as a fracture. He had great pain and was unable to use his leg. He remained in bed a month, and then managed to creep around on crutches. The pain in the hip continued, especially becoming worse if any weight was borne on the injured limb. Examination showed one and one-quarter inches' shortening on the injured side. The trochanter projected over an inch beyond Nélaton's line. By manual traction the shortening could be partly overcome and there was a sensation of crepitus. The radiogram (Fig. 10) revealed a fracture of the neck of the femur with absorption of almost the entire neck, and no union. Here we have a case in a young man in which three months after fracture the entire neck of the femur was absorbed, with resulting non-union. My experience has been that age has little if any bearing on the matter of union in cases of fracture of the neck of the femur. I have seen fractures in patients between sixty and eighty unite in the usual time with good functional results. Most of the cases of non-union that I have seen have been in persons of middle life. They were vigorous, healthy men, in whom the mal-union could not be ascribed to disease or malnutrition. It is a matter of local nutrition entirely. Fractures at the base of the neck are most liable to absorption of the neck, and fractures near the head are not so prone to absorption of the neck, and unite more readily regardless of the age of the patient.

In this case the same typical bone-peg operation was performed (Fig. 11). The graft was taken from the tibia. I will say that the fibula lends itself admirably to the same purpose. You need simply

to resect a suitable section and denude it of its periosteum. This man was in a body cast three months before any attempts were made to walk. Weight was borne gently on the leg after four months. Now, after the lapse of one year, you observe the result. He has no pain and practically no disability. He walks freely, with scarcely a limp. He climbs stairs normally, and I will now ask him to step on this chair with his injured limb. He is able to do it very well, and this I consider a good test of function. The result here is better than in the other patient. Not because there was less absorption of the neck; if any thing it was more complete, but this man was operated three months after injury and the other man was operated two years after injury. After two years the cicatrization and fibrous shortening of the tissues about the fracture make it impossible to correct the shortening as well as is the case three months after fracture. This experience would teach us to advise early operation. That is to say, in recent cases of three to four months' standing, with absorption of the neck and non-union, there is nothing gained by further delay in the hope of ultimate union occurring. As soon as the condition is assuredly permanent, then treat by operation. Going back further—in all fresh cases where the radiogram shows a fracture at the base of the neck, the possibility of non-union from absorption should be borne in mind. This should be taken into consideration in making a prognosis.

Who knows whether it may not some day be deemed justifiable to operate these cases at once, thus saving the patient a long period of convalescence and assuring him a good functional result. Such an operation in the hands of a competent surgeon is not difficult, and why should it not be done early in this instance as well as in the case of fractures of long bones? The next time a patient comes in my care with a fracture at the base of the neck—if he is young or middle age, and there are no contra-indications, I shall explain the situation to him and advise early operation. Why should we not in this way secure a perfect cosmetic and functional result, and save the patient a long period of disability?

OPENING CLINIC IN ORTHOPÆDIC SURGERY, TO THE SENIOR CLASS, STANFORD MEDICAL SCHOOL

By LEONARD W. ELY

San Francisco

I. COLLES' FRACTURE

III. EXOSTOSIS

II. BONE GROWTH

IV. TUBERCULOSIS OF HIP

ONE of our recent graduates told me the other day that he had gone out from the medical school with his diagnostic faculties well developed, but with vague ideas on treatment. Of course, if you can make a diagnosis, you are well on your way to proper treatment, but, on the other hand, when you graduate you should have some definite ideas on treatment, and during the time you are with me, I propose to give them to you. These ideas will often differ materially from those given you by your other instructors, and you must choose as best you can from the conflicting views. This is the function of a university medical school—to teach you to use your brains, not to administer mental pabulum whose constituents have been already agreed upon by the members of the faculty. If you want the evidence on which I base my views, it is always at your disposal. Much of it is in bottles and on slides in the laboratory.

When I studied medicine, we used to sit on benches, row after row, and listen to some old chap delivering the accumulation of the wisdom of ages down in the pit, but during our course you will be right at my elbow in the clinic, and if I put anything over on you it is your own fault.

Learn to pay a certain respect to authority. Don't be cocky. Accept tentatively what you are told until you can find something better, but if all the authority in the world is behind a theory and you know it to be wrong, do not cease to combat it. This is the joy of research, not only to find out something new, but to upset authority. The man with a sympathetic manner, who makes \$50,000 a year in his practice, never tastes it.

CASE I.—The first case to-day is one of an injured wrist. The

patient is a woman sixty-three years old, who fell down three steps four days ago, and injured her right wrist. She says she fell forward, with her right arm under her. She demonstrates the fall as well as she can.

She is quite sure she fell forward, and when she turned over, her right wrist was quite painful, and she has been disabled ever since.

You see that her right wrist is swollen, and slightly discolored. We note that she very carefully holds her right wrist steadied with her left hand, and that when we release the wrist she immediately resumes her hold. The right hand is abducted and the lower end of the ulna is prominent, constituting what is erroneously known as dislocation of the ulna. A suggestion of the well-known silver fork deformity of the forearm is present, but only a suggestion. If we had not made up our minds already as to the diagnosis, we possibly should not note any silver-fork deformity.

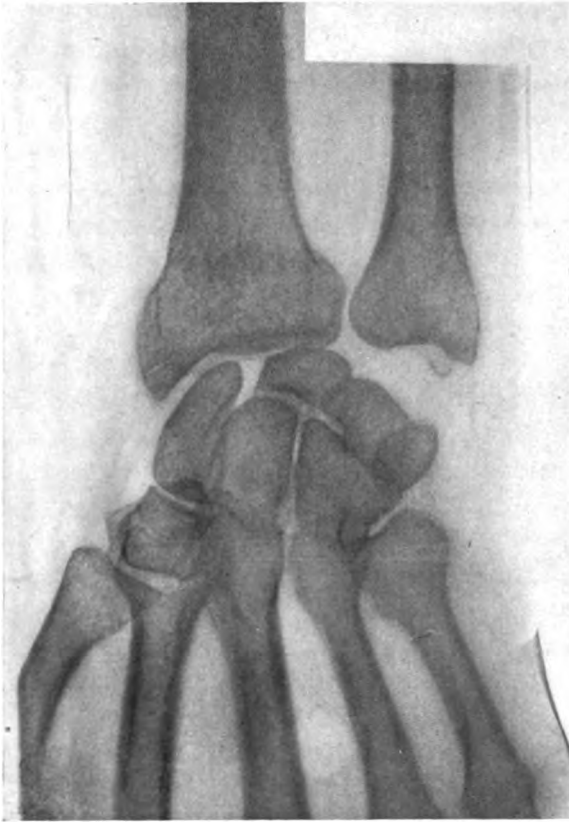
The hand is in pronation. Supination is slightly limited. Ecchymosis is present on the anterior surface of the wrist. Pronation is also slightly limited. Abduction, you see, is not limited. Adduction is slightly limited. Extension and flexion are both markedly limited, and all these motions are painful at their extremes.

When we come to the question of local sensitiveness, we find that the patient has marked sensitiveness over the lower end of the radius about three-quarters of an inch from the wrist-joint, and also over the styloid process of the ulna. No crepitus is present, and no false point of motion.

From the history of the injury, the resulting disability, the deformity, the ecchymosis, the swelling, the limitation of motion in the forearm, and especially from the distinct localized sensitiveness over the lower end of the radius, we can safely make a diagnosis of fracture of the lower end of the radius, and the pictures that she has had taken confirm this diagnosis, and show also that she has broken off the styloid process of the ulna. In the X-ray plates, you will notice that the antero-posterior view shows very little change, except in the styloid process of the ulna, but the lateral view shows marked displacement of the lower end of the radius, with a tilting backward of the lower fragment, and its impaction by the upper fragment (Fig. 2).

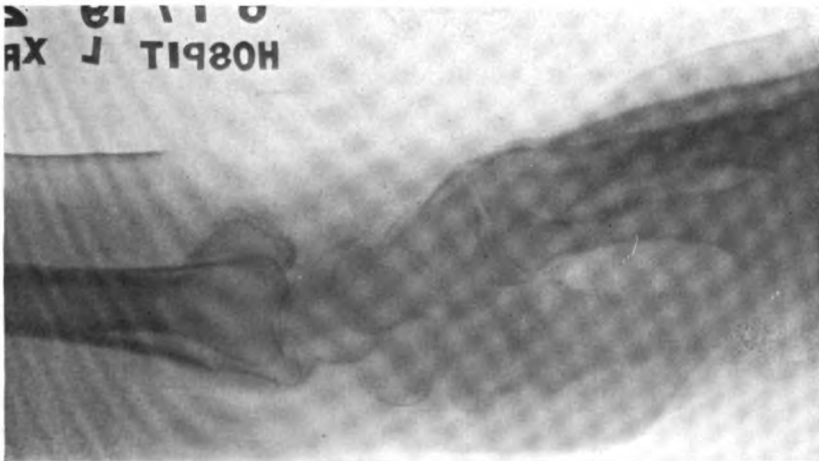
This fracture has occasioned a great deal of discussion. No two people quite agree as to all the facts in the case, and in any medical

FIG. 1.



Case I. Antero-posterior view.

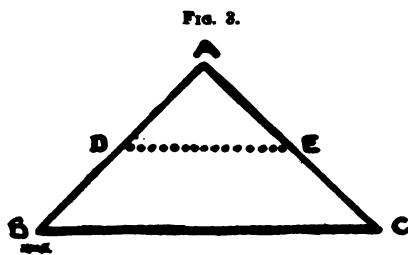
FIG. 2.



Case I. Lateral view, showing dorsal displacement of distal fragment.

meeting a heated argument can always be had by anyone who has anything to say on the subject.

For our purposes, we call a Colles' fracture any fracture of the lower end of the radius, with or without fracture of the styloid of the ulna, with or without impaction or comminution. The cause is usually a fall upon the hand; that is, by indirect violence. The same result can be obtained by the kick of a crank of a motor car. The patients always say that they fell forward. This patient states that she fell forward. As a rule, the patient's feet slip out from under him, and he falls straight down, and catches his weight on the heel of the superextended hand. Is the fracture then produced by the superextension of the wrist *per se*, or is it produced by the force exerted directly upward by the carpus on the radius? It can be produced on the cadaver in either way. The point is how is it ordinarily produced? I believe that the former is the mechanism in cranking a car, and the later invariably when the patient falls.



If a crushing force is applied at D. E. at right angles to the plane A B C, the apex D A E will yield much more readily than will the base and sides D B C E.

Why does the lower fragment tilt backward?

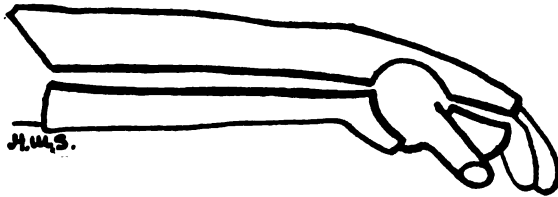
If you will saw through the lower end of the radius at the customary site of Colles' fracture you will find that the bone is triangular, with the apex of the triangle posterior. The force of the blow is transmitted by the carpus to the radius on a line parallel to the base and practically midway between the apex of the triangle and the base. The apex of the triangle, of course, yields more readily than does the broad base, and the impaction of the apex represents the giving way of the radius at its weakest part. (See Fig. 3.)

The diagnosis, as a rule, is easy. Sprains of the wrist are rare, are not very painful or disabling, and have limitation of motion in one direction only.

In fractures of the carpal bones, the sensitiveness is more distal, and the disability and deformity are not so great. In fracture of the navicular, with dislocation of the semilunar, the disability and deformity may be marked, but the sensitiveness is more distal than in Colles' fracture. This lesion may give a silver-fork deformity, and after the lapse of some time its appearance is practically identical with that of old Colles' fracture, and its diagnosis can be made only with the Röntgen rays.

The prognosis of the case depends usually upon the treatment. If the fracture is a simple one, as it is in this case, without comminution of the lower fragment, the result is excellent under good treatment. If the lower fragment is comminuted, the result is usually not so good. Once in a while under the very best treatment the result will be poor. Occasionally, under the worst treatment or with no treatment at all, the result will be excellent, but in the majority of cases the result is dependent on the treatment.

FIG. 4.



Plaster of Paris gauntlet.

The main idea of treatment is to break up the impaction under an anæsthetic, and then simply to hold the fracture until it is well. The form of splint is a matter of taste. Wooden splints, tin splints, cardboard splints, adhesive strapping, elaborate splints bearing authors' names—all have been used with success. Personally, I prefer plaster of Paris in the form of a gauntlet reaching from the elbow to the metacarpo-phalangeal joints, leaving the fingers and thumb free (Fig. 4). While the dressing is on, the patient is directed to use his fingers and thumb as much as possible.

The splint stays on for one, two, three, four, five or six weeks, as you wish. The length of time is of little importance if you can keep the patient from injuring the hand by another fall. Bony union is very rapid, so rapid that if treatment is delayed for a week the reduction of the deformity will be very difficult, or impossible.

Do not fear ankylosis as a result of immobilization. Fix your attention upon correct reduction of the fragments. I always put the hand up in pronation and slight flexion. Others believe in supination. Some adopt superextension. Always employ an anæsthetic. Do not attempt to reduce the fracture without one. Usually you will be allowed but one chance. If the patient absolutely refuses to take an anæsthetic, do not attempt to treat him unless he puts his refusal in writing, and signs it.

Slight prominence of the ulna and slight restriction of motion, in a woman of this age are to be expected. There is another point which I wish you to remember, and that is that people with bad teeth often have much disability after this fracture. Probably the fracture sets free into the joint the infectious material that previously was locked up in the marrow of the lower end of the radius, and a chronic infectious arthritis results.

We will now administer gas to the patient, and break up the impaction. The easiest way to do this is to grasp one fragment in each hand, and to move the two backward and forward on each other, using your knee for a fulcrum if necessary, persisting until you feel the fragments move on each other. Sometimes forced superextension will be of service.

You will see that the impaction is reduced, and that the deformity can be made to appear and reappear at will. The silver-fork deformity has gone and the forearm is straightened.

Will you all come down and feel this crepitus? I want to impress on your minds the importance of this. Until you elicit crepitus, the fracture is not reduced.

Having reduced the deformity, and having convinced myself that I have immobilized the lower fragment, I apply plaster of Paris, leaving the thumb and fingers free, and instruct the patient to use her fingers in every way that she can. I regulate the position of the hand while the plaster is setting. You see the plaster is quite soft, and while it is setting you can control the deformity to suit yourself. This is the great advantage of a plaster dressing. See how easily one can regulate it while the plaster is still setting. We could not do that with a wooden splint. We now trim the plaster, so as completely to free the fingers and thumb, and then, because the patient is going home, we slit the entire dressing from top to bottom, and warn her, if the

fingers become numb, or blue, or cold, to consult some medical man immediately. Otherwise, she will return for inspection to-morrow. Once more! *Break up the impaction under an anæsthetic and leave the fingers free.*

CASE II.—This patient is a woman thirty-four years of age, by occupation a housewife, who complains of pain in her right wrist. About three weeks ago while she was stepping into a store to do some shopping, she slipped and fell forward on her right hand and injured her wrist. She is sure she fell forward. All these patients are sure of it.

On inspection you will note that the right wrist is slightly swollen. Is that right? Is there anything characteristic about the swelling, or anything peculiar about it? (Answer): "No." That's right. It is simply a swollen wrist.

This patient exemplifies several of the things we discovered in our last case. In the first place, she says that she fell forward and shows how she fell forward with the hand in flexion. Manifestly it is impossible for any person to fall forward and land as she says she did.

When we come to examine the wrist we find that there is some swelling. All motions of the wrist are limited except possibly abduction. You will note slight sensitiveness of the lower end of the radius about three-quarters of an inch above the joint line. Running our fingers along the line on the dorsum we find there is a slight irregularity of the cortex in the sense that there is a sinking in about three-quarters of an inch above the joint line.

Three weeks ago a fall upon the hand, limitation of motion practically in all directions and great sensitiveness three-quarters of an inch above the joint line.

While a sprain can give limitation of motion, it gives it only in one direction, and then only when the torn ligament is put upon the stretch. Again, a torn ligament cannot give sensitiveness to pressure three-quarters of an inch above the joint line. Again, a sprained wrist from a fall is a rare thing.

The patient has excellent function in the fingers and slight limitation of all motions at their extremes, but both these are improving. Occasionally a case of Colles' fracture, no matter how well treated it may be, will get well with considerable impairment of function. Occasionally, as I mentioned above, a very bad fracture, without any

treatment, will recover with marked deformity but excellent function. This is one of the cases of moderate severity that will recover with fairly good function without treatment.

The X-ray picture shows a fracture of the lower end of the radius, and slight dorsal displacement with impaction (Fig. 5).

As the articular surface of the radius looks directly downward instead of downward and forward, the patient will have a slight disability, but owing to the three weeks' lapse of time since the injury, it hardly seems wise to administer an anæsthetic with the idea of correcting the deformity. We should probably fail. Union is rapid in this fracture. Whatever is done should be done within a week or ten days. After that an open operation is necessary, and an open operation is not indicated here.

As a rule, people with a sprained wrist do not consult a doctor. The history of a fall with resulting disability, and marked sensitiveness over the lower end of the radius, with or without deformity, usually means fracture.

CASE III.—The next case brings up bone formation, and here again we encounter a subject on which few people agree. The point is to get a theory of bone formation that will explain the results which men have obtained by animal experiments, without necessarily admitting the truth of their deductions, and also will explain what we see in the laboratory. This is an extremely hard thing to do. One man performs an experiment and reaches a certain conclusion, and another performs an experiment and reaches a conclusion quite the opposite. When we come to investigate the matter we find that the disagreement is due to two things: first, a lack of exact definition, and, second, a conclusion not justified by the premises—a case of *non sequitur*.

For instance, if MacEwen strips the periosteum off a bone and applies a ring of metal around the cortex, sews the periosteum over this ring, and finds that new bone is not formed on the outside of the ring, he is not justified in his assumption that periosteum has no function in bone formation. Bone is not formed on the outside of the ring because there is no necessity for bone there.

Again, if an experimenter buries periosteum in the soft tissues, and finds that bone forms in and on it, he is not justified in assuming that the periosteum "forms" bone.

If a hair-pin be lost in the bladder and becomes encrusted with calcium oxalate crystals we cannot say the hair-pin forms calcium oxalate.

What, in the first place, is marrow and what is periosteum? Ollier maintained that periosteum consists of two layers, an inner or cellular or cambium layer, and an outer or fibrous layer. The inner layer, he said, forms bone.

MacEwen says that the periosteum is a limiting fibrous membrane, and that it has no function in bone formation. Are there two layers? How can we find out? By looking at the periosteum under a microscope. Sometimes we will see two layers, but more often we will not, and if we cannot see them they are not there.

Periosteum is the tissue which covers bone in every place except at the site of the articular cartilage. It consists chiefly of fibrous tissue, but it may consist of fibro-cartilage, or cartilage. Its situation determines its name, not its structure. Prolongations of the periosteum run down into the bone, but these prolongations cannot be periosteum after they enter the bone, by the very definition of the term periosteum. They are marrow.

Marrow is the soft tissue within the bone—all soft tissue within the bone. Typical marrow tissue contains many cells of various kinds (the characteristic marrow cells), blood-vessels, fibrous reticulum, fat, etc., but again, the characteristic cells may be absent, and marrow may consist of little else but fibrous tissue or fat. It is none the less marrow. Like the periosteum, its situation determines the name of marrow, not the structure.

Pockets of typical marrow tissue can be seen on the outside of the cortex, but this cannot be marrow, though it has the typical structure. Inasmuch as it lies outside the bone, it must be periosteum. We have, then, typical periosteal tissue inside the bone, which is marrow, and typical marrow tissue outside the bone, which is periosteum.

With this definition of marrow and periosteum established, let us get together the accepted facts on bone formation, and see if we can build up our theory.

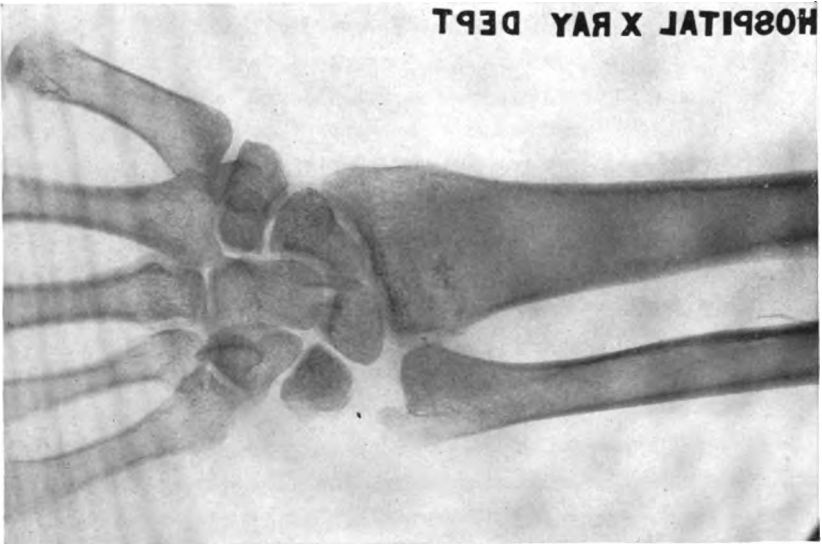
1. Intracartilaginous bone formation begins with the pushing of blood-vessels into the cylinder of cartilage—in a long bone. A similar process takes place later within the epiphysis. Here, then, is bone formation without marrow or periosteum.

FIG. 5.



Case II. Lateral view. Note that the lower articular surface of the radius looks downward, instead of downward and forward.

FIG. 6.



Case II. Antero-posterior view.

2. Bone is formed in the walls of the aorta, and in the kidneys of animals whose renal vessels have been tied off. Again, bone formation without marrow or periosteum.

3. Bone tissue is formed out of fibrous tissue or cartilage, within the bone, where no periosteum is.

4. Bone is formed in fibrous tissue—intramembraneous ossification—without cartilage, and before marrow is present.

5. Bone is formed on the outside of the cortex, beneath the periosteum.

6. Bone is formed in necrotic lymph-nodes and in the kidneys.

7. Bone contacted with bone and enjoying function, persists. Bone buried in the soft tissues, without function, slowly disappears.

To these I add another fact, whose truth I can demonstrate to you by stained slides.

8. Bone is formed in the fibrous tissue of the periosteum, not alone on the cortex under it.

Let me sum up:

Three things are necessary for bone formation.

1. Blood-vessels.

2. Either (a) a loose-meshed fibrous tissue, or (b) a homogeneous (cartilage matrix), or necrotic or granular material.

3. A stimulus, physiological or pathological, as the case may be. It is this stimulus that causes the blood-vessels to push into the cylinder of cartilage, in the first place, and that causes the bone production in the aorta. I know this is vague, but we can go no further at present.

You see, therefore, that neither periosteum nor marrow is necessary for bone formation, and that neither of them "forms" bone in the proper meaning of the word. In each tissue the conditions are right for bone formation. The materials are there, and given the stimulus, physiological or chemical, bone will be manufactured out of the fibrous tissue of the periosteum or out of the fibrous or cartilaginous tissue in the marrow. The presence of bone is to a certain extent a stimulus for further bone production. The true marrow cells, the characteristic marrow cells, have probably no bone-forming function whatever. The fibrous tissue of the periosteum is like other fibrous tissue of the same consistency. It forms an excellent building material.

In your search for truth, don't elaborate a theory from your inner consciousness and then start out to demonstrate it by animal experimentation, for you will always succeed. Assemble all your facts first, and then draw your deductions.

The blood-vessels are the active element, but what is the essential individual cell that forms bone? There is such a cell if the prevailing theory of bone formation—the neoblastic theory—is correct. It is called an osteoblast. Some say the osteoblast is a mesoblastic cell brought by the blood-vessels. Moschcowitz says it is simply an endothelial cell from the blood-vessel wall. It possesses no physical characteristics by which we recognize it. It is recognized simply by its situation on the margins of the bone trabeculae and on the bone cortex. When we see small round, or polyhedral cells on the margins of the trabeculae we say that bone is being formed, but here we are liable to err. We often see a similar picture when the bone is being torn down.

Let us see of what value all this is in considering our next patient.

This patient is a salesman, recently a soldier, nineteen years of age, who comes in and tells us that he has an "osteoma" on the inner side of his knee. He has ridden horseback more or less for the past two years. Inspection shows a swelling on the medial aspect of the right thigh. Palpation reveals this as a hard mass, not fluctuating, and not connected with the skin—apparently connected with the femur. It is slightly sensitive. The overlying tissues are not inflamed. Pressure elicits a peculiar crepitus over the proximal part of the swelling.

The X-ray shows a bony spur arising from the region of the adductor tubercle of the femur, about four centimetres long, consisting of dense bone at its base and spongy bone at its end—resembling in appearance the appendix. This is what is known as "riders bone." It occurs almost invariably in men who ride horseback a great deal. Evidently, then, the element of irritation is the exciting cause. Similar spurs and exostoses—non-malignant—occur in other parts of the body, notably on the tubercle of the calcaneus. Their favorite sites are at or near the attachment of muscles and near the epiphyseal cartilages. If they cause trouble they should be removed. Sometimes they are multiple with unknown cause, but with perhaps an hereditary element. These we let alone, as long as they are painless.

Sometimes these growths consist partially of cartilage. They may be covered with it more or less completely—*exostosis cartilaginea*.

Our incision here, about twelve centimetres long, reveals the posterior margin of the vastus medialis. We retract this forward and show the spur, covered with a loose periosteum, and apparently with irregular islands of cartilage at its extremity, but not connected with muscle or tendon. We divide the periosteum at its base, so as to remove as far as possible material for further production, and chisel the spur off flush with the bone.

Will the spur recur? We do not know, but to be on the safe side, we remove a small piece of fascia from the edge of the wound, and sew it over the bare bone. If we did not do this and had not removed the periosteum with the spur, and if the patient continued to ride horseback, we should have, of course, practically everything in favor of further bone production; namely, the bare bony base with its marrow containing blood-vessels—the builder, the periosteum—the building material, and the occupation—the stimulus.

We now suture the wound in layers, and apply a dry dressing.

When you remove a piece of bone do not regard it as a solid, useless thing, and throw it away; but, as a shell containing perhaps the most interesting, active, changeable, complex tissue in the body, preserve and study it. You will gain in this way a better comprehension of bone disease than you can in any other, and will change a routine procedure into an incident in an absorbing occupation.

CASE IV.—The patient is a girl seventeen years of age, who first came to the clinic March 18, 1919; that is, about four months ago, complaining of limp, and pain in her right lower extremity. One year previous she commenced to be lame in her right hip. No history of a fall can be elicited. Three weeks before we saw her, while getting out of a crowded street car, she was accidentally "bumped" on her knee by someone in the street, and thereafter suffered an increase of pain in the knee. The knee became a little swollen.

There are nine children in the family altogether; one died of endocarditis. The patient has always been in health. Measles in infancy. Scarlet fever at twelve years. No pneumonia. Pertussis at three.

The patient walked at that time only with assistance. The lower

extremity was in flexion. Motion in all directions was markedly limited, with muscular spasm.

Measurement showed no shortening of the lower extremity. The right thigh was smaller, showing atrophy of one inch, and the right calf one-half of an inch. The hip was at an angle of 140° and there was practically no flexion possible. There had been no history of sore throat. The teeth were in shocking condition.

The Röntgenogram taken three months ago showed that the head of the bone was not clearly outlined, and that there was a rounded area of decreased density in it. The Röntgen diagnosis read "possible tuberculosis of the right hip." Manifestly at that time the patient had arthritis of the hip.

The next question is: What sort of an arthritis was it?

The changes that we see in the femoral head are those that can be caused by a number of organisms, and these are the tubercle bacillus, the treponema pallidum, and the diplo-streptococcus, especially that form of this last organism that is found in diseased tonsils, or in the deep genito-urinary tract. There was no reason to suspect in this case either lues or diseased tonsils. From the fact that the patient was young, that the disease had lasted about one year, had slowly but steadily progressed, and had never involved any other joint, the supposition was that it was tuberculosis. On the other hand, the mouth condition made one suspicious of an infection from the teeth, but the form of arthritis that goes with diseased teeth is not this form of arthritis. Almost invariably arthritis that follows infections of the jaw is of the second type, the type with bone production at the margins of the joint, with wearing away of the cartilage and eburnation—our Type II, the German arthritis deformans, the English osteoarthritis, Goldthwait's hypertrophic arthritis, Nichols and Richardson's degenerative arthritis, etc. Again, the second type of arthritis is not so likely to be as painful as this evidently was. However, with marked dental infection, our course was plain, and we had the mouth put in order. Abscesses were found at the roots of the teeth. The next indication was to reduce the deformity and put the joint at rest. Whatever the cause of arthritis, rest will probably cause an amelioration of the symptoms, and in the case of a tuberculosis, we shall be administering the best treatment possible. In other words, we stand

to win something and lose nothing. If the joint is syphilitic, the process will not be affected one way or another with rest.

Under ether we reduced the deformity, and put the hip in a plaster of Paris spica from the waist to the knee.

The patient improved, the pain slowly disappeared. She got about on crutches and was fairly comfortable. She reported at the clinic regularly and we noticed that she steadily improved. One week ago we removed the plaster, and found the hip in an excellent attitude of abduction and slight flexion.

When you put a hip in plaster of Paris and reduce the deformity, with a possibility of ankylosis, always put it in abduction and full extension. Then, if ankylosis result, the hip is in the best functional attitude.

The patient was sent to the X-ray room and we had the second picture taken, which I show you. The report reads as follows:

"Marked destruction of femur and of acetabulum, being a very marked extension of the process of destruction over previous examination. Diagnosis, therefore, tuberculosis."

A steadily progressive, destructive process in the bone marrow, causing rarefaction of bone, and irregularity of the joint surface, evident in both the acetabulum and in the head of the femur, which has increased in spite of the fact that we have had it put in plaster of Paris, and has never involved any other joint, without other discernible cause, enables us to make a tentative diagnosis of tuberculosis, but not a positive one. This can be done only by the demonstration of tuberculosis under the microscope.

The changes in the bone in tuberculosis are the same as those in lues, and we must never leave that possibility out of consideration. Therefore, a Wassermann test was done, and the patient was placed on iodide of potassium and mercury. The Wassermann test is negative.

With the diagnosis fairly positive, what shall the treatment be? We can continue to put the patient in plaster of Paris, and we can assure the parents that after the end of three or four years we shall *perhaps* succeed in getting a stiff, almost painless hip in good attitude, with a possibility of recurrence of the disease at any time by a twist or wrench.

We can tell them, on the other hand, that with an operation we can probably cure the disease and enable the patient to go about

with a limp, but without pain, at the end of a few months. If we operate, we shall operate with only one idea in our minds, and that will be to destroy function in the joint—to destroy the joint as a joint. That is not yet established authority, but if you will think of the disease as I have shown it to you in the laboratory, you will see that it is the only rational theory on which we can operate. Manifestly one cannot remove all the diseased tissue. The process is not only in the femoral head and in the soft tissues, but in the acetabulum as well. The pelvis is involved to a degree which we are unable to estimate, before the operation or during it.

Some maintain that if in your operation you remove a certain amount of diseased material, nature will “take care” of the rest. That is absurd. Nature has been unable to take care of the original tubercle in the bone marrow or synovial membrane. How, then, will she take care of the large amount you will leave behind, when you add the insult of a severe operation? Nature’s efforts are all being extended in the direction of destroying the joint. If we amputate, we still leave the diseased pelvis behind.

There is but one logical theory of operating on a tuberculous joint, and that is to imitate Nature, and to do thoroughly what she usually is unable to do—to destroy function in the joint.

The presence of tuberculosis in the bone and joint is due to the presence there of two tissues vulnerable to the tubercle bacillus, the synovial membrane and the lymphoid marrow. Except in children, the presence of these two tissues in a general way may be said to be dependent on function. If function be removed these two tissues disappear. If they disappear tuberculosis disappears—it is starved out. In other words, no function, no synovial membrane and lymphoid marrow; no synovial membrane and lymphoid marrow, no tuberculosis.

This rule presupposes one thing and has one great exception. There are some cases which are doomed from the beginning. Nature has no resisting power against the disease. Even an amputation will be followed shortly by the death of the patient from tuberculosis of some other organ. A resection will not heal. You have seen under the microscope specimens from such cases, and have had the chance to compare them with others.

We add one other cardinal principal—avoid secondary infection. In the presence of secondary infection tissues previously immune to

the tubercle bacillus become vulnerable, and for a simple, comparatively harmless, strictly localized disease, a widespread and very dangerous one is substituted.

Therefore, in all operations on tuberculosis joints, never open and drain, never scrape and pack, with the idea of providing exit for the tuberculous material. You will, on the contrary, provide entrance for pus germs. Plan your operation to destroy the joint, and to secure primary union of the wound.

In this case we have a choice of procedures: (1) To remove the head of the femur, and cause a dislocation upward. (2) To do an Albee operation and secure bony ankylosis. In the first the stump of the femur would be slung by a thickened capsule on the dorsum of the ilium, with a movable thigh, and a shortening of about two inches. In the second the hip would be stiff, but not shortened appreciably.

I choose the first, and proceed to the operation. The incision runs distally from the anterior superior spine for about sixteen centimetres. Dissection carries it down to the capsule. Before we reach it we encounter, as you see, a large amount of flocculent fluid. Evidently this has burst through the capsule from the hip-joint, and keeps welling up as we proceed. Probably we have evacuated six or eight ounces—typical tuberculous pus—a cold abscess. We open the capsule, and find the head badly diseased. We remove it with a few blows of the hammer and chisel, scrape out the acetabulum, remove all detritus, sew up the wound, and put on our sterile dressing. While my assistant applies a plaster spica from nipples to toe, with the hip in extension and abduction, I will pass the femoral head around for your inspection.

Bring up in your minds the destructive inflammation which has existed in the bone marrow, and you will understand the damage that has been done in the bone and cartilage of the specimen—the rarefaction and death of the bone, and leafing off and death of the cartilage. Serum and white blood corpuscles are added to this, and a cold abscess is formed in the joint, which bursts through the capsule.

If we can secure primary union of our wound, we shall cure the joint tuberculosis.

HEREDO-SYPHILIS IN THE THIRD GENERATION WITH NO STOMATA IN THE SECOND

FROM THE MEDICAL CLINIC OF THE PETER BENT BRIGHAM HOSPITAL, BOSTON, MASS.

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UNTIL the last decade a great deal has been written concerning the transmission of both the virulent and the dystrophic types of syphilis to the third generation. So many cases with essential details have been reported and so much evidence founded on scientific basis has been offered that the most skeptical individual accepts it as an established fact. The paucity of literature by American writers is noticeable and during the time that the study of syphilis has been well controlled by serological study, very little has been written, either by foreign or American writers.

As stated by Marshall¹ it seems to have been generally accepted that in order to prove the transmission of syphilis to the third generation, the following rules should be fulfilled:

1. The presence of acquired syphilis in one of the grandparents (first generation).
2. The presence of undoubted heredo-syphilis in one of the parents (second generation).
3. The presence of heredo-syphilis in the child (third generation).
4. The absence of acquired syphilis in either parent.
5. The absence of reinfection of the heredo-syphilitic mother with syphilis.
6. The absence of the intervention of another syphilitic genitor.
7. The absence of acquired syphilis in the child.

Boeck,² Fournier,³ Goucher, Taylor⁴ and others have reported many cases which fulfill all of the above requirements.

The above postulates were made before our study of this subject was controlled by the Wassermann reaction. From more recent investigation it appears that they are too rigid and that the condition

may exist when not all of these postulates are fulfilled. The following cases are of this type:

CASE I.—J. R. I., age seven, was seen October 24, 1913 (Med. No. 572, Peter Bent Brigham Hospital).

The child was brought to the hospital complaining of sleeplessness and restlessness. In the fall of 1911 the mother first noticed that the child slept very poorly. At that time he became very nervous and the condition gradually grew worse. In September, 1911, about ten days after a fall down a flight of stairs, he developed a sudden excruciating headache which persisted several days and associated with this were nausea, vomiting and fever. After that time until his entrance to the hospital he appeared to have a constant dull headache and every two or three months he would have severe paroxysms of pain in his head lasting two or three days. Nausea was not a prominent symptom, but following some of the paroxysms of pain in his head he would vomit nearly constantly for three or four days. Beginning March, 1911, the child's temperature was taken four times a day by his mother. She reported that his temperature was constantly elevated (99° to 100°). February, 1912, he developed drooling from the mouth, which gradually grew worse. About June, 1912, the mother first noticed that the patient's speech was getting slow and hesitating. Pronunciation at that time was good and ideas not confused. The impairment of speech gradually became worse. About this time he began to stumble and would often fall. He began to take less notice of things about him. Objects in the distance did not attract his attention at all. At this time he developed a slight weakness in his right leg and a definite disturbance in gait was noticed. He was taken to the Johns Hopkins Hospital and while there had a constantly elevated temperature. Difficulty in hearing was then noticed. In December, 1912, he stopped telling his mother when he wished to go to the toilet and from that time on he was incontinent. His mother was uncertain whether he could not control his sphincters or whether his mental condition was at fault. In January, 1913, he became unable to speak. He was able to recognize only gross objects. The following February the right arm and leg showed definite evidence of being weaker than the left but at no time was he paralyzed in any extremity. After August, 1913, he was unable to walk unaided. His mother now noticed that he could not drink from a glass and

that he seemed to make no effort to swallow. The dysphagia gradually increased.

The father is alive and well. He denies venereal disease. Blood Wassermann done at the Peter Bent Brigham Hospital was negative. The mother is alive and well. She denies lues and her blood Wassermann done at the Peter Bent Brigham Hospital was negative. She had a miscarriage in 1901, and another in 1905. Two children, now fifteen and twenty years of age, respectively, were then born. They are healthy and apparently perfectly normal. No family history of tuberculosis or nervous trouble with the exception that the maternal grandfather died of "locomotor ataxia" was obtained.

The child was a full-term and healthy baby. Forceps were required at delivery. He was breast fed. He talked at the end of a year and walked at the end of seventeen months. He was always considered healthy before the onset of the present illness. No sores or rash had ever appeared on his body. He had the earache at three or four years of age which left a subsequent discharge. He had frequent falls in babyhood. In 1910 he was tossed in a blanket and allowed to fall on the floor, striking his head, but he was not unconscious, neither did he bleed from the nose, mouth or ears. He fell down a flight of twenty steps in 1911 following which he became nervous and developed his first severe headache.

Previous Treatment.—In March, 1912, the patient was seen by a number of physicians who made a diagnosis of tuberculosis of the brain. He was examined by others in September, 1912, and was considered as a case of tuberculosis meningitis. He was taken to Johns Hopkins Hospital in November, 1912, where he was under the care of Drs. G. J. Hoyer, H. M. Thomas and Adolph Meyer. The Wassermann reaction done with his blood and spinal fluid was found positive. At that time he was given salvarsan and again on December 30, 1912, and in February, 1913. At this time his blood and spinal fluid Wassermann reactions were still positive. He was given a course of iodide and mercury, but his condition was reported worse on discharge.

Physical Examination.—October, 1913, the child was considerably under size for his age of seven. The Hutchinson triad was not present. The circumference of the head was 50.5 cm. There was slight asymmetry in the parietal region as the right eminence was unquestionably more prominent than the left. The temporary teeth

were in bad condition. The central permanent incisors were saw-toothed but not pegged. The lungs were clear and the heart was normal. The arm and leg measurements were within normal limits. The testicles were in the lower canal but easily pushed into the scrotum. The penis was normal.

Neurological Examination.—*Subjective.*—Sleeplessness and restlessness since 1910. Disturbance of speech since 1912. He had had severe paroxysms of headache, beginning January, 1912. His gait became affected in 1912. After December, 1912, he was incontinent to urine and feces. He could not walk unaided after August, 1913. There was slight weakness of the right arm and leg. His mentality was definitely impaired during the two years previous to his entry. *Objective:* He is left-handed. The right parietal eminence is larger than the left. His mentality is much below par. His orientation to time, place and person is entirely absent. There is complete sensory aphasia. There is slight muscle weakness of the right arm and hand. A positive Rhomberg is present. He is unable to walk without assistance. His superficial reflexes are not elicited. Bicep reflex is slightly greater on the right. The knee-jerks are equal but markedly exaggerated. Oppenheim, Babinski and Gordon signs are absent. He is incontinent of urine and feces.

Röntgen Examination on October 28, 1913.—There is a small sella. The whole skull has some hydrocephalic configuration. On side view the whole parietal and temporal regions show definite thinning of the bone. There are no signs of increased intracranial pressure.

The white blood count is slightly elevated.

Operation.—On October 30, 1913, an exploration of the right hemisphere was done by Dr. Harvey Cushing. His notes show that the dura appeared normal but just beneath it was a grayish semi-transparent membrane covering the entire hemisphere. Beneath this there could be separated another thin membrane. The thickness of the three was about 3 mm. The brain was greatly atrophic. The entire dura was removed and the bone flap was replaced over the dural window. Doctor Cushing's diagnosis was a syphilitic pachymeningitis. The hemisphere was well sopped with 1-5000 bichloride of mercury, but the Wassermann reaction done three weeks later showed no change from the positive character. Smears made at the time

were stained with Giesma and examined by Doctor Councilman for the spirochæte, but were reported negative.

The *post-operative history* was not remarkable. The wound healed perfectly. Previous to his operation and after the immediate reaction from the operation, he ran a fever ranging from 99° to 100°. A diagnosis of cerebral syphilis and chronic pachymeningitis was made. On February 22, 1914, he was transferred to the medical service for further observation and treatment. His condition had not improved. He remained on the medical service one week, at which time he was discharged untreated and unimproved.

CASE II.—Mrs. A. K. (Peter Bent Brigham Hospital, O. D. D. No. 57641, A. K. 41706; E. K. 57788.) I shall give only a brief report of this case.

A woman recently entered the Out-patient Department of the Peter Bent Brigham Hospital complaining of pain in the right hip of one year's duration. She was the eldest child of a family of four. She was forty-four years of age. While living in South Africa, 16 years ago, she married a German. She had never been married before and denied any chance of acquiring syphilis either accidentally or in the usual way. Her husband admitted of having had gonorrhœa previous to their marriage but denied syphilis. His blood Wassermann examination was negative. One year after marriage the patient gave birth to a normal child—A. K., who did not have snuffles, was nursed by her mother, and with the exception of being undersized and having scapulæ that are suggestively scaphoid, I am unable to find anything about the child which is abnormal. Three years later the patient gave birth to a second daughter, who is also underdeveloped. On physical examination I was unable to detect any definite abnormality except for a possible mitral regurgitation. Four years later a third daughter was born and her physical examination reveals no variation from the normal. The mother of these children has had no miscarriage or still-births. No children are dead. The father of the mother developed "locomotor ataxia" at forty-five years of age and died a few years later. Except for the mother's present complaint she has been healthy. Her blood Wassermann reaction has been persistently a plus minus to a single plus, which represents in our system of reporting Wassermann reactions 25 per cent. to 50 per cent. inhibition. An X-ray of the hip shows a distinct flatten-

ing of the head of the femur. Anti-syphilitic treatment has greatly improved the condition.

Blood taken from the fifteen-year-old daughter shows a persistent single plus Wassermann reaction, while blood from the twelve-year-old girl shows a repeatedly double plus. The third girl's blood gives a negative Wassermann reaction.

There are several other similar cases on our records which are just as conclusive as these.

The first case reported does not fulfill all of the rules generally accepted for establishing heredo-syphilis in the third generation. The maternal grandfather had "locomotor ataxia." Neither parent, in the second generation, showed any stigmata of heredo-syphilis. The child as noted by the history and physical examination had stigmata that could be due to heredo-syphilis. Both parents denied venereal infection and the blood Wassermann reaction on both was negative. The question of an intervening genitor was inquired into and, according to the parents' statements, was ruled out. Of course, it is impossible to absolutely rule out this possibility. The mother and father were asked concerning the various possibilities of the child acquiring syphilis accidentally. They were intelligent and observing parents and could give no history that would lead one to believe that the disease was acquired.

In the second case there is a history that the maternal grandfather died of "locomotor ataxia." Neither parent showed any of the ordinary evidences of heredo-syphilis. The mother at forty-four years of age is having a painful hip. The X-ray shows definite bony change. Anti-luetic treatment has relieved the symptoms. Her blood Wassermann has been repeatedly slightly positive. The father's blood is negative. Neither of the three children shows any definite heredo-syphilitic stigmata with the exception of being undersized and having somewhat of an elderly appearance. The blood Wassermann reaction of the first two daughters has been repeatedly positive while that of the youngest has been negative. The question of syphilis in the mother, acquired either accidentally or in the ordinary way, has been carefully inquired into, and no history can be obtained that would indicate it. So far as an intervening genitor is concerned, it does not seem probable that the first two daughters would receive their positive Wassermann reactions from that source, neither does it seem

possible that both daughters of this age would have acquired their syphilitic condition.

In considering the first case, does it not seem possible that a child with inherited syphilis may be born from parents whose physical and serological examinations are normal? Does it not seem possible that virulent spirochaetes may be encapsulated in some of the tissues of the body, such as the ovary, either without antibodies in the blood stream or antibodies not in sufficient quantity to be detected by our most delicate serological tests, and yet under certain physiological or pathological conditions these may be transmitted to the offspring? This would explain many of the positive Wassermann reactions obtained in children and young adults, the source of which we are unable to determine.

The second case impresses one with the importance of not considering too lightly a persistent slightly positive Wassermann reaction, even when obtained by the strongest antigens, if well controlled. The blood of the mother showed 25 per cent. to 50 per cent. inhibition. Evidently there are only a few antibodies in her blood stream, yet her offspring has blood whose Wassermann reaction shows a 100 per cent. inhibition.

Although the obstacles in actually proving the transmission of syphilis to the third generation are many, nevertheless since these cases have been carefully studied from the standpoint of history, physical and serological examination, it seems most probable that their grandfathers were the original source of their inherited syphilis. If these cases are acceptable, the transmission of heredo-syphilis to the third generation is not the rarity it has been generally considered to be.

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Medicine

THE PRESENT STATUS OF BLOOD-PRESSURE

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FROM time to time we must pause in our efforts to improve our methods of diagnosis by instrumental aids and seek to evaluate the collected results lest we be carried away by enthusiastic advocates, or held back by skeptical reactionaries. Unfortunately, every method of procedure advanced by a man, or group of men, is made to appear at first to be the very last word on the subject. No account is taken by the inventors of many variabilities of the human organism in health and disease. Laws are formulated on insufficient data, which have a tendency to pass into medical literature as dogma, and are believed in by many as are the tenets of a religion. The diagnostic help afforded by estimation of the blood-pressure has been no exception to this well-known but oft neglected rule.

To discuss a subject it must be defined. Blood-pressure is the force used by the heart to keep the blood in equilibric circulation. In order that the heart may be able to perform this function it must have a left ventricular muscle more or less capable of strong and uniform contractions. The distributing channels (the aorta and large branches) should be elastic and the arterioles should have intact muscle walls. Further, the vasomotor sympathetic system should be in good tone, and the venous return flow from the capillaries to the heart should be unobstructed. Let us bear in mind all these accessory factors, for changes in any one, or more, may modify the blood-pressure.

Let us suppose a normal adult heart in good function. At the beginning of systole all the valves are closed. The blood in the body is being forced along to the capillaries by the elastic rebound of the

large arteries. The intraventricular pressure rises rapidly until it is just greater than the pressure at the aortic valve on the aortic side, when the aortic valves open and 100 c.c. or more of blood are rapidly propelled into an aorta fairly well filled with blood and still contracting upon the contained column of blood. The heart contracts until it empties itself, the diastole begins, the aortic (pulmonic) valves close, the mitral (tricuspid) valves open. The blood is kept circulating during diastole by the elastic recoil of the aorta and large branches. Just as much blood pours into the ventricles during diastole from the great veins as is forced out during systole. This is elementary physiology, but often forgotten.

There are three terms used in blood pressure. The maximum or systolic pressure represents the full force of the cardiac contraction. The minimum or diastolic pressure is the pressure at the aortic valve during diastole. It represents the actual contractile force which the left ventricle exerts before one drop of blood enters the circulation. It is, therefore, supposed to measure the peripheral resistance, as this is the back pressure which keeps the aortic valves tightly closed. It is possible that this is true, because in states of low peripheral tension the diastolic pressure is found to be low. The pulse pressure is the difference between the maximum and minimum pressure, and, therefore, actually measures the pressure under which the blood is forced along the great vessels towards the periphery. It is evident, therefore, that only a part of the actual contractile force of the left ventricle is concerned in the movement of blood to the periphery.

The systolic, the diastolic, and the pulse pressure taken together represent what I have called the pressure picture. The whole must be taken in order to gain information of value in blood-pressure determinations. One might just as well count the red blood corpuscles and name the kind of anemia present as to take the systolic pressure and expect to gain valuable information concerning the circulation. It is true that much has been learned by taking the systolic pressure alone, as much may be learned by counting the red cells alone. Because this is so is, however, no argument in favor of continuing a poor method of gaining information.

Yet another factor in blood-pressure must be taken into consideration. That is the venous pressure. Wherever there is engorgement there is obstruction to flow and increased resistance, which is reflected

back upon the arterial side. This necessarily affects the blood-pressure. Venous return, then, must be free in order to insure even blood-pressure and perfect circulation.

It is assumed that the auscultatory method of taking blood-pressure is being used and that the reader is familiar with the five phases in the average normal person. The first phase represented by a short sharp click; the second phase by a click and murmur; the third phase by a loud, clear, snappy tone; the fourth phase by a dull tone, which quickly dies away to no sound, the fifth phase. All these phases can be easily heard by gradually lowering the air pressure in the arm band while listening over the brachial artery at the bend of the elbow. There are a few precautions which should always be observed in order to take a reading.

1. The patient should be resting in a comfortable position with all muscles relaxed.

2. The patient should have rested at least ten minutes after entering the physician's office.

3. The patient should be mentally relaxed.

4. The cuff should be snugly, but not tightly, and smoothly wrapped around the bare arm and the cuff should be warm.

5. The lower end of the cuff should not come to the bend of the elbow.

6. The arm should not be fully extended, except when the patient is lying in bed.

7. The bell of the stethoscope should be evenly placed over the lower end of the brachial artery about 2 cm. from the lower end of the cuff. The artery should bisect the circle made by the bell. The bell should not be pressed too firmly, nor should it be tilted so that it partially occludes the lumen of the artery, either proximally or distally to the center of the bell.

8. All parts of the instrument should be airtight.

9. Pressure should rapidly be made above systolic pressure. The air then may be rapidly reduced at the first reading in order to determine roughly the upper and lower limits of sound.

10. At the second reading the pressure in the cuff is then raised to a point about 10 mm. above maximum pressure and the air valve opened so that the pressure in the cuff falls slowly.

11. The first two readings should be discarded, as they do not

represent the true pressure. The first one is always much too high (except in rare cases), the second one is usually too high.

The first sharp tone which is definitely heard over the artery as the pressure in the cuff is released is the systolic pressure. It is usually a short, quick tone. Sewall has recently called attention to the well-known fact that the respiratory waves modify the tone. That is to say, if the pressure in the cuff is held at the first audible tone, the tone comes and goes with respiration, and not until the pressure in the cuff has fallen appreciably more are all heart beats heard as sharp clicks. This is true. The deeper the respirations the greater difference there is between the highest point where the tone is first heard and the point where all tones (heart beats) are heard. He raises the question, Where is systolic pressure to be measured? In dyspnoea this would be difficult. Personally, I should take the average of the high point where an occasional tone is heard at expiration and the point where all tones come through. For the average patient during quiet respiration there is practically so little difference due to the respiration that it can be disregarded entirely.

The diastolic pressure is read at the point where the loud tone (the so-called third tone) suddenly becomes dull. Here also there is often, especially in high tension, a difference of 20 mm. between that point and the disappearance of all sound. In cases of aortic insufficiency there is always a dull sound heard over the uncompressed artery. I have shown both clinically and experimentally that the place to read the diastolic pressure is at the change from loud third to dull fourth tone. This is now generally conceded. Practically, it makes little difference in the average case whether one reads the diastolic pressure there, or at the disappearance of all sound. The difference amounts to from 4 to 8 mm. It is only necessary to remember that in the interpretation of the reading. The pulse pressure can now be determined. In general it is about 40-45 mm. for an adult. Many a man goes about with a pulse pressure of 30 mm. and 20 mm., which is not incompatible with life, provided there is not increased pressure. Above 60 mm. is certainly to be classed as high pulse-pressure.

What are the normal systolic and diastolic pressures? In spite of the great numbers of estimations made, I do not believe there are any figures for normal pressure at any age, or for either sex. There

are average pressures present in healthy persons. These may be said to be between 120–180 mm. Hg. systolic and 80–90 mm. Hg. diastolic, giving a pulse-pressure which has limits of 30 to 50 mm. Hg.

After all, nothing is gained by attempting to read to the millimetre. What difference does it make if the systolic is 122 or 132 mm.? It is normal. If the diastolic is 88 or 76 mm.? It is normal. Entirely too much attention has been focussed upon the actual instrumental reading. There are many sources of error in any clinical instrumental method. Not alone is the factor of the observer to be reckoned with, but the many variables in human beings must be borne in mind. If it is the first time the patient has ever had the blood-pressure taken, up goes his systolic pressure. If he happens to take a deep breath just as the pressure in the cuff is nearing systolic pressure, one reads too high a systolic pressure. Should he be excited, nervous, or have just taken a hearty meal his systolic is higher than when he is quiet, or between meals. I have long ceased to be interested in mere figures to represent the blood-pressure, except in a general way. I care not whether the systolic is 180 or 220 mm. It is high. That is the important feature. But how about the diastolic pressure? That is, in my opinion, the more important of the two extremes. However, I wish to emphasize the point that the whole pressure picture is important. The diastolic pressure measures dead weight, wasted energy, if you like, and so a diastolic which approaches the average systolic is a sign that the heart is doing a great amount of unnecessary work. The diastolic pressure is, furthermore, not influenced by the causes which produce changes in the systolic pressure. It is, therefore, steadier under all conditions, and when it is low, or high, it has really some significance in itself.

To lose sight of the fact that the blood-pressure is only one link in the chain of evidence which leads to diagnosis is to miss the whole point of the value of blood-pressure estimations. Evidently many have been guilty of this blindness. The fault has lain in those of us who, in order to press a point, have stressed one feature of the diagnosis. Now that blood-pressure is a recognized procedure, it behooves us to correct our mistake. I think it can be said that there are certain conditions where blood-pressure readings are diagnostic principally in a negative sense. For example, a normal average pressure picture is incompatible with a chronic interstitial, or chronic diffuse nephritis.

I have found average normal in cases of chronic parenchymatous nephritis (the small white kidney with few interstitial changes). Again, a normal systolic and very low diastolic cannot occur in any cardiac valvular lesion except aortic regurgitation.

In spite of experimental work showing that intracranial pressure raises blood-pressure, there is little clinical evidence to support the observation. Intense grades of cerebral compression occur without any appreciable change in the blood-pressure, so that the estimation of pressure is of little value in diagnosis.

In all fevers the blood-pressure picture tends at first to show an increased pulse-pressure. The rate of the heart is increased and the diastolic pressure, due to vasodilatation, tends to assume a lower level than in health. In exhaustive fevers the systolic later falls so that the pulse pressure may be as low as 20 mm. Hg. This is scarcely enough to force blood to the brain in the sitting, or erect posture, so that dizziness, or fainting results in those who attempt too early to assume these positions.

In pneumonia it has been held by some that the danger point to the circulation is reached when the heart rate reaches, or passes, the number of millimetres of systolic pressure. Probably this is true for some persons; it certainly is not true for all persons. If the average systolic were known of all those who developed pneumonia some reliable data might be assembled. The fact that this is not known renders conclusions based upon such an arbitrary formula of no great value.

Hypotension; that is, a blood-pressure picture below normal, has, so far as we know, no great significance. Many exceedingly healthy persons live an active life with a systolic pressure of 110, or lower, and a diastolic pressure of between 70 and 80 mm. In such persons exercise raises the systolic normally, as it should, without affecting greatly the diastolic pressure, and the recovery is normal. Hypotension is seen often in old people with senile arteriosclerosis when the myocardium is the seat of atrophy and increased pigmentation. Hypotension is also seen in Addison's disease, in advanced carcinoma, advanced tuberculosis, and other chronic diseases.

In shock and hemorrhage the blood-pressure falls rapidly, the systolic always more rapidly than the diastolic, so that the pulse-pressure becomes low and in fatal cases rapidly approaches and reaches

zero. Before that stage arrives it is not possible to measure the blood-pressure, as the patient is pulseless.

Hypertension (increase in the blood-pressure picture) is the feature of blood-pressure which has claimed the most attention. Hypertension, I believe, is a conservative and compensatory process. It is the response on the part of the circulatory system to keep the body going in the face of factors which are tending to destroy it. No one will deny that an extreme grade of hypotension is in itself an element of danger to the afflicted person and must be looked upon as one of the factors in prognosis. It is, however, my belief that hypertension does not begin as a disease in itself, but as the result of conditions with many of which we are unfamiliar, but which cause the heart and circulation to respond with increased pressure in order to keep the circulation in equilibrium. Weigert's law of overgrowth holds for every tissue in the body. The general tendency of all tissue when injured badly is to heal with an increased amount of tissue which eventually become fibrous. Other tissues which are injured can be put more or less completely at rest. Consider for a moment the enormous callus thrown around the fractured ends of bone where there is too much motion allowed during healing.

The heart and arteries cannot be put at rest during the time when poisons, of whatever nature, are acting upon them. However, there is overgrowth in the form of enlarged and increased muscle fibres in the heart, yet eventually fibrous tissue is deposited and muscular force is diminished. The fact that a vicious circle is finally established and the fact that states of great hypertension in themselves are harmful do not seem to me to prove that hypertension is not a conservative process.

We see acute and chronic hypertension, or better, subacute and chronic. Then there are persons who appear to have hypertension as a normal state. The anomaly seems to be in families and to be hereditary. Such persons are always stocky, thick-necked, red-faced, with solid fat. No kidney lesion can be found and no functional change can be demonstrated by any test. The knowledge of the hypertension is usually gained at a Life Insurance Examination or accidentally by having the pressure tried as a stunt.

Entirely too much stress has been laid upon hypertension. The laity become panicky when told that the blood-pressure is high. It is

time that a halt was called to the flood of loose statements published broadcast and guzzled down by poorly trained doctors. The men who write have a great responsibility resting upon them which some appear to take lightly. We have frightened the public nearly to death. Let us attempt to inculcate a sane attitude towards this subject. The sphygmomanometer has come to stay. Practically every doctor carries one in his bag. The patient asks why the doctor did not take the blood-pressure.

Again, hypertension in itself is not dangerous and should not be treated by vasodilation drugs. We are too prone to think that hypertension has the patient rather than that the patient has hypertension. It is axiomatic that the part cannot be as great as the whole.

Hypertension is an indication of some process going on in the body which results in increased work for the heart. At times it is temporary, or subacute, and apparently due to poisons generated in the alimentary tract or connected with some endocrine gland dysfunction. Such a condition as the hypertension of the menopause belongs to the endocrine gland pathology. Again, the rising blood-pressure in pregnancy is often only temporary, but is a most valuable indication of probable disease of the kidneys and of probable eclampsia. No obstetrician does his full duty to the pregnant woman if he fails to take the blood-pressure picture frequently during the last half of her pregnancy. Hypertension and chronic interstitial or chronic diffuse nephritis are not synonymous. There are at least three types of chronic hypertension: (1) the chronic interstitial nephritic; (2) the essential or hereditary; (3) the arteriosclerotic. There are grades of all three and combinations of any two, so that it is difficult often to place the individual who has hypertension in any one group. Since 1917, when I published this grouping, I have not seen any better classification. I have found it of value, and, therefore, await a better classification before discarding it.

*"Group 1 (Chronic Nephritis).—*These are the cases with a high-pressure picture; that is to say, high systolic (200+) and high diastolic (120–140+). The pulse-pressure is much increased. The palpable arteries are hard and fibrous. There is puffiness of the under-eyelids, which is more pronounced in the morning on arising. Polyuria with low specific gravity and nycturia are present. There are almost

constant traces of albumin in the urine, with hyaline and finely granular casts.

"Functionally these kidneys are much under normal. The functional capacity determined by Mosenthal's modification of the Schlayer-Hedinger method shows a marked inability to concentrate salts and nitrogen. The phthalein output is below normal. As the case advances the phthalein output becomes less and less, until a period is reached when there are only traces or complete suppression at the end of a two-hour period. Such patients may live for ten weeks (one of our cases), all the time showing mild uræmic symptoms, and suddenly pass into coma and die.

"The natural end of patients in this group is either uræmia or cardiac decompensation (so-called cardiorenal disease). Cerebral accidents may happen to a small number. It is only to this group, in my opinion, that the term of cardiorenal disease should be applied. Formerly I believed that all high systolic pressure cases were cases of chronic nephritis of some definite degree. From the purely pathological standpoint that is true, but from the real, the functional standpoint, it is far from being the true state of the cases.

"In this group there is marked hypertrophy and moderate dilatation of the left ventricle, with dilatation and nodular sclerosis of the aorta. The kidneys are firm, red, small, coarsely granular, the cortex much reduced, the capsule adherent. Cysts are common. It is the familiar primary contracted kidney. Mallory calls this capsular-glomerulo-nephritis. The etiology is obscure. Often no cause can be found. Again, there is a history of some kidney involvement following one of the acute infectious diseases, or it may follow the nephritis of pregnancy. Usually, however, these cases fall into the group of secondary contracted kidneys, chronic parenchymatous nephritis."

"*Group 2.*—This one might designate as the hereditary type, although there is not always a history in the antecedent. This group includes the robust, florid, exuberantly healthy people. They often are heard to boast that they have never had a doctor in their lives. They are usually thick-set, or very large, fleshy people. The pressure picture is exceedingly high. The pulse-pressure is moderately increased. The arteries are rather large, fibrous, and often quite tortuous, although this is not always the case. Some persons have hard,

small, fibrous arteries. There is no puffiness beneath the eyes, no polyuria, and no nycturia as a rule. The urine is of normal amount, color, and specific gravity. Albumin is only rarely found, and then in traces, but careful search of a centrifuged specimen invariably reveals a few hyaline casts. The phthalein excretion is normal or only slightly reduced. The kidneys excrete salt and nitrogen normally. It is in this group that apoplexy is found most frequently. The rupture of the vessel occurs when the victim is in perfect health, often without any warning. Occasionally when such a case recovers sufficiently to be around, cardiac decompensation sets in later, and he dies then of the cardiac complications.

"Pathologically the hearts of such persons are found to have the most enormous hypertrophy of the wall of the left ventricle. The cavity is somewhat enlarged, as is always the case when the pulse-pressure is increased, but the size of the cavity is not the striking feature. The aorta is fibrous, thick-walled, and the arch is slightly dilated. There are patches of arteriosclerosis. One such case seen only at autopsy had a rupture of the aorta just above the sinus of Valsalva and died of hemopericardium. The kidneys are of normal size, dark red, firm, the capsule strips readily, the surface is smooth or finely granular, the cortex is not decreased. The pyramids are congested, and red streaks extend into the cortex. Microscopically the capsules of the glomeruli are a trifle thickened; a few show hyaline changes. There is rather diffuse, mild, round-cell infiltration between the tubules. The tubular epithelium shows little or no demonstrable changes. The arterioles are generally the seat of a moderate thickening of the intima and media, but it is not usual to find obliterating endarteritis. There is evidently a diffuse fibrous change which has not affected either the tubules or glomeruli to any great extent."

"*Group 3.*—This might be called the arteriosclerotic high-tension group. The cases are usually over fifty years old. They are men and women who have lived high and thought hard. Often they have had periods of great mental strain. Many men in this group were athletes in their young manhood. Many have been fairly heavy drinkers, although never drinking to excess. They are usually well nourished and inclined to stoutness. The pressure picture is high systolic, with normal or only slightly increased diastolic and large

pulse pressure. The arteries are large, full, fibrous, usually tortuous. The heart is very large, the apex far down and out. There is no polyuria; nycturia is uncommon, quite the exception. The urine is normal in color, amount, and specific gravity. Albumin is only rarely found and hyaline casts are not invariably present. The phthalein excretion is quite normal and the excretion of salt and nitrogen are also normal. The terminal condition in most of the patients in this group is cardiac decompensation. They may have several attacks from which they recover, but after every attack the succeeding one is produced by less exertion than the preceding one, and it becomes more and more difficult to control attacks. Eventually the patients become bed- or chair-ridden, and finally die of acute dilatation of the heart.

"Occasionally patients in this group may have a cerebral attack, but in my experience this is uncommon. Pathologically the heart is large, at times true *cor bovinum*, dilated and hypertrophied. The cavity of the left ventricle is much dilated. The aorta is dilated and sclerosed.

"The kidneys are increased in size, are firm, dark red in color, with fatty streaks in the cortex. The capsule strips readily and the cortex is normal in thickness or only slightly increased. The organ offers some resistance to the knife. The microscope shows small areas scattered throughout where the glomeruli are hyalinized, the stroma full of small round cells, the tubules dilated, and the cells are almost bare of protoplasm. Naturally the tubules are full of granular cast material. Also the arterioles show extensive intimal thickening, fibrous in character, with occasional obliterating endarteritis. One gets the impression that the small sclerotic lesions are the result of anæmia and gradual replacement of scattered glomeruli by fibrous tissue. For the most part the kidney, except for the chronic passive congestion, appears quite normal. One can readily understand that in such a kidney function could not have been interfered with."

Hypertension has always been understood tacitly to mean increased systolic pressure. With this implied conception I heartily disagree. The systolic pressure is only one end of the blood-pressure; the other end is just as, if not more, important. Who will say that a man with systolic pressure of 160 mm. and diastolic pressure of 120 mm. has only possibly a mild hypertension? To me that is much

more hypertension than the man who has 180 mm. systolic and 90 mm. diastolic.

There are at least two disturbing elements in the former case. In the first place, while the pulse-pressure is apparently normal, the heart has to exert force up to average normal systolic pressure before blood enters the aorta. So much is wasted energy. Under such circumstances the circulation is poorly maintained. In fact, such a hypothetical pressure indicates impending cardiac failure. In the second place, all cases of compensated chronic hypertension have left ventricles which are hypertrophied and which have also increased blood capacity. If the heart is acting well all the blood in the ventricle is propelled into the aorta at every systole. Therefore more blood is forced into the aorta, there is a greater stretching of its walls and a greater volume of blood is forced onward. Increased pulse pressure and increased cardiac output go hand-in-hand. Compensated hypertension is only possible with increased blood-pressure, which means increased output, and is associated with enlarged ventricular capacity.

If this be true, and there is evidence at the autopsy table to prove it, then the pulse pressure becomes a most important figure to know. This can only be obtained by reading the diastolic pressure.

To take the first example again, 160 mm. is not high in a man of sixty years. Suppose that was the only figure taken. Would there not be a grave omission and a consequent error in prognosis and treatment? Or suppose the systolic pressure were 180 or 200 mm.. would it not be most valuable to know whether the diastolic pressure were 130 or 100 mm.? My contention is that it is so valuable that only by obtaining the pressure picture are we enabled to give intelligent prognosis.

Over two years ago I said, "No blood-pressure reading is complete unless both systolic and diastolic pressures are determined.

"Pulse pressure is important because it gives the actual driving (kinetic) force which keeps the blood in motion, and has, therefore, a great value in prognosis.

"High pulse pressure is invariably accompanied by four conditions, some of which can always be determined, and frequently all of which can be found. These are (1) increased size of the cavity of the left ventricle; (2) definite dilatation of the arch of the aorta; (3)

bronchial or tubular breath sounds heard over the manubrium, and (4) increase in size of all distributing arteries."

Arteriosclerosis and hypertension are not necessarily coincident. Frequently they are. The large, fibrous, diffusely thickened artery and the relatively small, hard, fibrous artery are associated with hypertension. On the other hand, the goose-quill or beaded, pipe-stem artery of senile arteriosclerosis is apt to be found with hypotension.

Finally, my conception of the present status of blood-pressure boiled down to its concentrated essence, is that blood-pressure has been considered an end not a means to an end. We must use it as one of many valuable diagnostic and prognostic aids. We must have the pressure picture before us in order to arrive at any reasonable conclusion. We must look upon hypertension as a symptom, not as a disease. Hypertension is only a physical, or physico-pathological, expression of something occurring in the body of a human being.

A little mental journey now and again into the foggy cerebral atmosphere where the fundamental sciences lie is excellent to clear the fog and to keep one from travelling jauntily along the speculative white way.

THE SIGNIFICANCE OF CARDIAC DULNESS AND METHODS OF USING IT *

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RADIOLOGICAL STANDARDS

ACCORDING to Claytor and Merrill's röntgenographic tables of normal heart-size in men standing, the transverse diameter varied from 10.7 to 14.0 cm., showing a difference of 3.3 cm. Groedel found differences of 3.4 cm. and Dietlen of 4.3 cm. These are the differences found in what seem to be the most reliable röntgenographic standards of the normal. The findings in individual cases which fall within these limits can only be judged with an approach to accuracy by taking into account the *body-weight*, the *shape of the chest* and *position of diaphragm*.¹¹ The influence of age, sex, stature and occupation may have to be considered also in some cases.

Having recognized the difficulty of appraising all these factors correctly, T. Groedel¹ in 1918 published a brief note on comparison of the transverse diameter of the heart with transverse internal diameter of the chest or lungs. If the former be indicated by "T" and the latter "L. T.," his findings can be stated as follows:

In children	T : L.T. = 1.0 : 1.90
At age of 20 yrs.	T : L.T. = 1.0 : 1.92
At age of 30 yrs.	T : L.T. = 1.0 : 1.95

In a subsequent paper, F. M. Groedel² says that when "T" is greater than one-half of "L. T." the heart may be considered enlarged. This cannot be meant exactly, for the limits given for normal are from 1.90 to 1.99.

This attempt by the Groedels to avoid using their own very extensive tables of heart-measurement seems particularly significant. If the radiologist can prefer a ratio to a tabulated standard, the clinician might do the same.

* From the Medical Out-patient Department of the Massachusetts General Hospital.

RELATIONS OF X-RAY TO PERCUSSION MEASUREMENTS

Radiological methods, being more accurate than percussion, have been used as a check on percussion. It is important, therefore, to know how they can best be applied for this purpose and whether or not measurements so obtained are directly or indirectly comparable with measurements of percussion outline.

If the chest were cylindrical and its wall of uniform structure, dulness caused by the proximity of the heart could be mapped out with considerable certainty, because the chest-wall would then modify the percussion note in a uniform manner. The reverse is true. The variations in structure of the chest-wall cause alterations of resonance which, on the right, render the recognition of *slight cardiac dulness* difficult and uncertain.

The situation in regard to the left border of the heart is quite different. Comparison of percussion outlines with teleoröntgenograms or with orthodiagrams has shown that the outer limit of cardiac dulness always lies to the left of the heart-border as indicated by the X-ray.

The reason is because the orthodiagraph projects a silhouette of the heart perpendicularly onto a flat surface in a single plane, whereas percussion is performed on a curved surface, upon which cardiac dulness, projected outward along diverging lines, becomes magnified in extent (Fig. 1). This is not an error of percussion. It results from conditions inherent in the structure percussed. Percussion consequently gives a magnified picture which is, in a measure, stereoscopic. Measurements of dulness obtained by simple methods of percussion, therefore, should not be compared directly with those obtained by means of the X-ray.

Outlines of the heart, however, have been made in such a way that they corresponded closely with the silhouette in a large proportion of cases. I refer to the work of Dietlen,³ Treupel,⁴ and others. Their outlines were obtained, not by simple and direct use of percussion, but by a complex system of interpreting the significance of different degrees of dulness. They used different criteria for different parts of the heart-border, so that the result represented an intricate series of deductions rather than a record of simple observation.

Of these men it may be said that they were particularly well

qualified to outline the heart, because of extensive experience with orthodiagraphic work; that, nevertheless, errors were occasionally considerable, that the conditions of quiet under which they worked are obtainable only with difficulty, and that to master their technic would require prolonged training checked by the X-ray. I have repeatedly succeeded in making on the chest-wall outlines of the heart which corresponded closely with those of the X-ray,¹⁰ but have had numerous failures. When successful, the result has been reached indirectly by palpating the apex impulse and by making allowance for past discrepancies rather than by straightforward use of percussion.

I believe that the attempt to outline the whole heart by percussion in such a way that it shall conform to orthodiagraphic standards adds a new difficulty to an already complex problem, that the results even in the hands of experts are likely to be less reliable than if based on simpler methods, and that it would be folly for one not experienced in the use of röntgenograms of the heart to base conclusions on findings by these methods of percussion.

If measurements of percussion outlines are not directly and simply comparable with orthodiagraphic standards of heart-size, is there a constant relation between them which might justify comparison? So far as known to the writer no such relation has been demonstrated.

The conclusion seems inevitable that radiological standards of *transverse diameter or of mid-sternum to left border* are not satisfactorily comparable to measurements derived from ordinary methods of percussion.

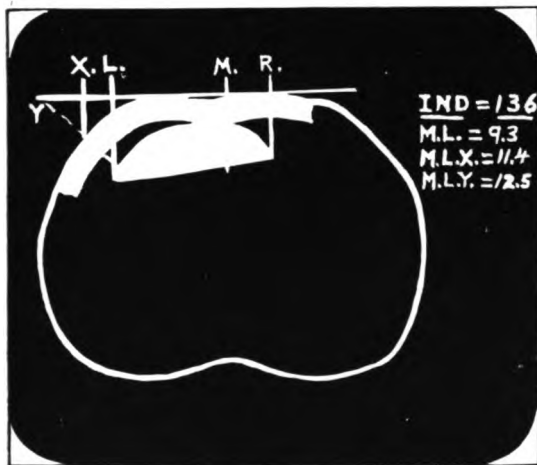
Although no constant relation has been shown between measurements from mid-sternum to *right border* of dulness and the corresponding measurement of the silhouette, comparison of these is justifiable as a check for percussion because the chest in this region is nearly flat.

The X-ray may and should be used to *check inferences derived from percussion*. When rightly employed it is of great value.

BASIS FOR MEASUREMENTS OF DULNESS

Before the X-ray came into general use there were few who attempted to outline the heart by percussion. Differences of teaching in regard to the normal outline of cardiac dulness in those days were

FIG. 1.



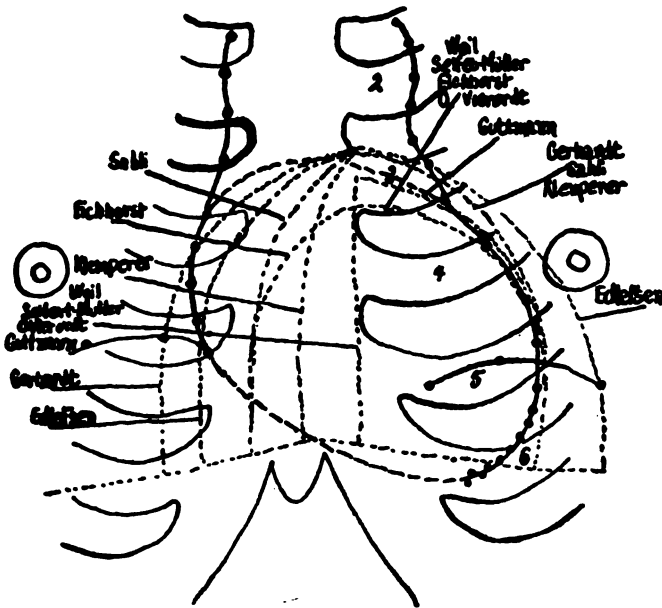
This diagram is constructed from measurements taken from a man having a chest of average shape and a heart showing no evidence of abnormality. It shows the relation of cardiac dulness by two methods of measurement to the radiological measurement (Shattuck).

illustrated by Moritz * (Fig. 2). There is less diversity of opinion to-day, but disagreement between examiners as to borders of dulness is not uncommon in particular cases.

Such disagreements are attributable partly to differences of method of percussion, and partly, it seems, to the subconscious influence of diverse conceptions of the normal position of the heart; but the greater differences of opinion are caused by the absence of any generally accepted standard for the *degree of dulness which should be recognized as representing the cardiac border*.

One examiner makes his mark where he finds the first slight diminution of resonance, another regards only considerable changes of resonance, and a third uses one standard for the right border and a different standard for the left border.

FIG. 2.



Outlines of normal cardiac dulness as depicted by various teachers and superimposed on the orthodiagram of a normal heart (Moritz).

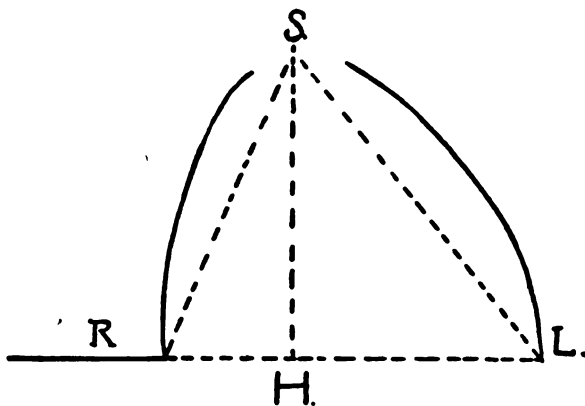
The borders of cardiac dulness are not sharply defined. Consequently, a measurement of cardiac dulness as determined by any individual has not the mathematical accuracy that the figure connotes. The figure does not represent a fact easily verifiable by means of an instrument of precision. What it really represents is mainly the

judgment or opinion of the examiner. Consequently, its value as evidence is little better than that of an opinion, although the form of statement makes it appear to have the significance of fact.

METHODS OF MEASURING DULNESS

In order that recorded measurements for cardiac dulness, as in case reports or hospital records, should be understandable, there must be no doubt as to how they were taken; and it would be advantageous to have them always made and recorded in the same way. Two methods of measurement are in common use for the left border. Some people use a flexible rule, bending it to conform to the curve of the chest. Others project an imaginary perpendicular from the mark on the skin to the coronal plane in front of the chest. Comparison of these

FIG. 3.



Norris and Landis' method of recording cardiac dulness.

methods shows that the distance from mid-sternum to left border of dulness is from 0.5 to 1.0 cm. greater by the first method than by the second. Advocates of the flexible rule object with justice to the perpendicular method on the ground that it is less easy to measure accurately. The others reply that measurements taken around the chest give an exaggerated idea of heart-size.

The following figures taken from a normal case with a chest of average shape show a sample of the relations of percussion measurement to each other and to X-ray findings, Fig. 1.

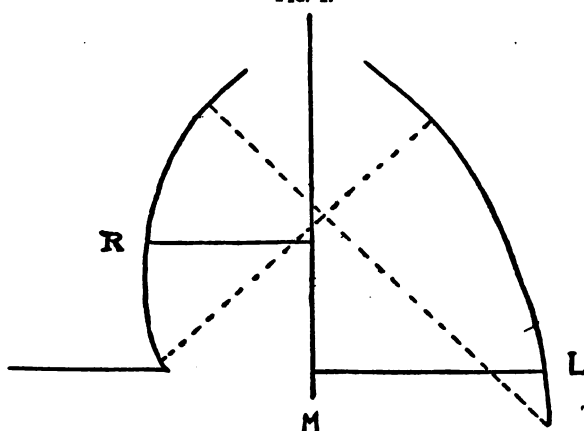
Mid-sternum to cardiac apex by X-ray (ML).....	9.3 cm.
Percussion outline measured from coronal plane (MLX) ..	11.4 cm.
Percussion outline measured around chest (MLY)	12.5 cm.

Another point emphasized by Norris and Landis⁵ seems important. They say that "the *area* of heart dulness is normally from 10 to 30 per cent. larger in the horizontal than in the erect posture." Measurements of transverse diameter do not vary to the same degree, but posture is taken into account by radiologists.

The following average measurements of transverse diameter, or "T," were obtained by radiological means:

Average for men standing (Clayton and Merrill) *	= 12.1 cm.
Average for men supine (Dietlen) *	= 13.2. cm.
Average for women standing (Claytor and Merrill) *	= 11.1. cm.
Average for women supine (Dietlen) *	= 12.1. cm.

FIG. 4.



Moritz's method of measuring cardiac dulness (Norris and Landis).

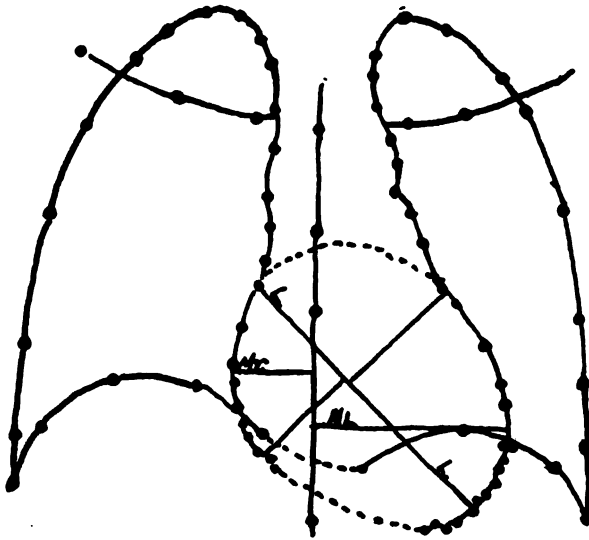
In some individuals the differences caused by change of posture are much greater. This is notably the case in persons having viscerop-tosis, a low diaphragm when standing and a "drop" heart. On lying down the diaphragm ascends and pushes the heart up so that in the supine posture the heart may assume the ordinary shape. Such a change of shape causes a very marked change in transverse diameter, and cardiac dulness should change correspondingly.

Two diagrammatic methods of recording measurements are in common use—that of Norris and Landis (Fig. 3), and that of Moritz (Fig. 4). I have not tried the former, but prefer the latter method because I percuss the right border in the fourth interspace and the left border in the fifth interspace. Liver dulness in the fifth interspace

on the right interferes with detection of cardiac dulness, and, moreover, in a heart of normal shape the right border extends farther to the right at a little distance above the liver than it does lower down. The method of Moritz is similar to that in common use for orthodiagrams (Fig. 5).

For the reasons stated it seems clear that measurements made by one examiner cannot safely be used by another unless their methods of obtaining and of recording measurements are the same. The

FIG. 5.



Orthodiagram of the heart showing the usual method of measurement (Dietlen).

reader, at any rate, should be informed how the measurements were obtained.

The possible value of diagrams or measurements made by an individual examiner for his own use in detecting changes occurring from time to time in the size of the heart of an individual patient is not questioned.

INTERPRETATION OF DULNESS

If it be granted for purposes of discussion, but contrary to fact, that methods of measurement are uniform, so that the figures can be

interpreted by anyone who reads the record, the next question is to what standard shall they be compared?

They might be compared with tables of orthodiagraphic measurement, but it was pointed out above that the two are not satisfactorily comparable.

Thayer⁷ says that the position of the apex impulse in normal individuals lies from 7 to 10 cm. to the left of the median line, and that percussion shows dulness about 1 cm. farther out. He finds the left border of dulness, therefore, from 8 to 11 cm. from the mid-sternum. He places the right border of dulness at from 2.5 to 4.5 cm. from the median line. If it is permissible to add together the minimal figures on the one hand and the maximal on the other, it appears that normal transverse diameter may vary from 10.5 to 15.5 cm., which allows a margin of 5 cm. for individual differences within normal limits. This does not signify that an originally small heart which has become enlarged can increase in diameter by 5 cm. before enlargement can be detected by means of percussion, but it follows that a skilful interpretation of measurements is essential before reaching a conclusion.

To interpret measurements of dulness in the individual case it is necessary to take into account the limitations and possible errors of the method employed, and also to consider the *shape of the chest*, the *probable position of the diaphragm*,¹¹ and the *body-weight* of the patient. It may be important in some cases to consider also size of chest, sex, age, stature and occupation of the individual before deciding whether the measurements of the heart are normal or abnormal.

If measurements of cardiac dulness are to be recorded as criteria of heart-size, the data above mentioned should always be recorded with them.

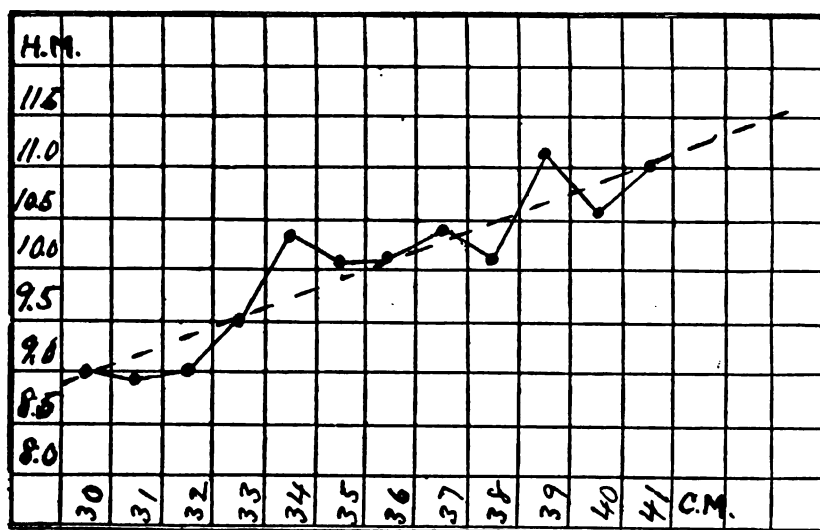
Difficulties such as these caused the Groedels^{1, 2} to turn aside from orthodiagraphic tables and to offer in their stead an index of heart-size based on the ratio between the transverse diameter of the heart and that of the lungs (see above). This method * is not yet fully developed, but seems likely to gain ground in the future. The suggestion follows naturally that if there were a satisfactory index for cardiac dulness, it would, at least, save trouble.

* Danzer¹² used the "cardiothoracic index" in a large series of cases. He believes it valuable, but makes reservations.

AN INDEX FOR PERCUSSION

Goodman and Harris⁸ have recently offered a plan which seems to have some advantages. They discard physical landmarks entirely, and claim that "measurement of the heart-boundary from the median line is the only reliable clinical method," but say that "to interpret these figures properly, data should be furnished as to body-weight and circumference of chest." Their figures show a fairly constant relation between chest-circumference and percussion measurement from mid-sternum to left border (Fig. 6). They found a somewhat

FIG. 6.



Figures in vertical column represent heart-measurement from median line to left border of dulness in centimeters, "H. M." The figures at the bottom are the circumference of the chest in inches, "C. M." The data is that of Goodman and Harris, adapted from their "Chart No. 8."

The relation of heart-dulness to chest-circumference is clearly shown.

similar relation between measurement of dulness and body-weight. The curves differ, however, in the higher figures where increase of heart-size does not keep pace with body-weight. It is known that the hearts of fat individuals are small in proportion to body-weight, so that failure of the curve of heart-measurement to rise proportionally to the higher weights might be explained on this ground. The relation of heart-measurement to chest-measurement appears to be more constant.

The question then arises whether body-weight could not be dispensed with, leaving the relation of heart-measurement to chest-circumference as the sole criterion. If a fairly constant ratio exists in all sorts of normal cases between heart-measurement and chest-measurement, it might be advantageous to use it alone instead of weight tables and complex methods of interpretation. The ratio between heart-measurement and chest-circumference in Fig. 6 was obtained by dividing the latter by the former. The ratio varies from 3.30, the lowest, to 3.77, the highest, and the average is 3.52. This ratio might serve as a normal heart-index.

Before making use of such an index it is necessary to consider more fully its limitations. Assuming that Goodman and Harris⁸ measurements were made under standardized conditions, the index should be applicable by using their methods under similar conditions. Its applicability might be limited, however, to cases of the sort examined by them. They worked only with men of military age undergoing military training. More varied material would, probably, have shown greater variations in the index. Their percussion measurements taken from mid-sternum to left border differed only from 9 to 10.5 cm., whereas Thayer⁷ found differences of from 8 to 11 cm. in similar measurements. Therefore, the limits of variation shown by this index will not serve as a criterion for Thayer's⁷ work. If an examiner desired to use such an index prepared by others, he might properly wish to know the standard posture and methods of percussion and of measurement upon which the index was based. If these methods differed much from his own, the index would probably be valueless to him, but he could provide a similar index from his own findings and make use of it in like manner. For example, if one should obtain minimal, maximal and mean or average figures for chest-circumference, and corresponding measurements from mid-sternum to left border, using a representative group of cases sufficient in number, an index could readily be prepared from them. It might or might not prove satisfactory in practice and would probably fail to indicate enlargement, mainly to the right. An index based on transverse diameter might be better.

In summing up the discussion of measurements, it may be said that transverse measurement and measurement from mid-sternum to

left border both show marked differences in individual cases; so that complex interpretation is required before the significance of the figures can be determined accurately. Therefore, he who makes use of measurements without fully appreciating the limitations and sources of error of his method is in danger of falling into grave error.

Another objection to measurements as criteria is that they give an appearance of accuracy and of simplicity which is fallacious. Unless rightly restricted, the use of measurements is unsound and consequently unscientific.

LANDMARKS AS STANDARDS

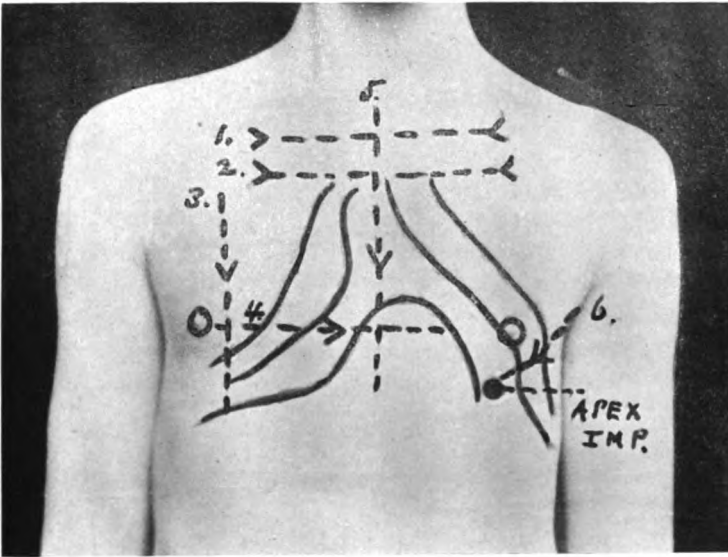
No simple standard exists with which measurements of cardiac dulness can be compared. While chaotic conditions relating to measurement of the heart prevail, and until some standard emerges which is worthy of general acceptance, the individual examiner seems most likely to get reliable results by adhering to that method of determining heart-size in which he is most proficient.

I prefer to judge of relative heart-size by comparing the findings of percussion with landmarks of the chest. Interspaces, the margins of the sternum, and the mid-clavicular, anterior axillary, and mid-axillary lines, are used.

Goodman and Harris⁸ condemn the use of the mid-clavicular line as an indication of heart-size. Their data seem to justify their conclusion that measurement to the mid-clavicular line bears a different relation to body-weight than does the heart-measurement from mid-sternum. The same is true of the relation found for the mid-clavicular line and heart-measurement as compared with chest-measurement. These authors have *not shown clearly the relation of the mid-clavicular line to the left border of dulness*. Their mean figures, whether classified on the basis of chest-circumference, of body-weight, or of stature, represent group-averages which make the desired direct comparison impossible. That figures for the two measurements differ is clear but not important.

The contention of Goodman and Harris⁸ that the mid-clavicular line is useless as a standard may be justified by figures which they possess, but the evidence presented for their statement is confusing and insufficient.

FIG. 7.



The continuous lines indicate, from without inward, very slight dullness, slight dullness and flatness in an individual case. Such outlines vary much in different normal individuals. Neither of the three outlines by itself bears a constant relation to heart-size.

The dotted lines indicate routine lines of percussion, the arrowheads the direction of the percussion, and the numbers the order in which each line is followed.

A METHOD OF INTERPRETING DULNESS

The method to be described was adopted five years ago after a series of comparisons between percussion outlines and teleoröntgenograms. The radiological work was done by the late Walter Dodd or by George W. Holmes, so that the accuracy of this part of the work, within the limits of error inherent in the method, can be taken for granted.

Before describing the method, my belief should be stated that any stroke, however light, if strong enough to elicit a clearly audible lung-note in a resonant part of the chest, will penetrate deeply enough to show comparative degrees of resonance in any region near the heart; and, further, it has been my experience that slight differences of resonance are more easily appreciated when the percussion stroke is very light than when it is heavier.

Heavy percussion is used only to obtain a rough idea of relations. Cardiac dulness is then studied with the lightest strokes which afford a clearly audible lung-note in a resonant part of the particular chest under examination. The patient is examined sitting. When dulness to the right of the sternum is ill-defined, the examination is repeated while the patient lies on the back.

No attempt is made to outline the heart. Instead of this the relations of very slight dulness, slight dulness, and of flatness to landmarks are noted (Fig. 7). The varying sense of resistance under the finger is used as an auxiliary means of detecting diminished resonance in the region of the heart.

The first step is to determine the limits of dulness in the region of the great vessels and the degree of dulness over them. Percussion is begun below the outer third of the right clavicle in the first interspace and carried across the sternum to a corresponding point on the opposite side; then back over the same line. The second interspace is then percussed in like manner (Fig. 7).

The second step is intended to give information about cardiac dulness on the right. There is generally so much dulness in the fifth interspace from other underlying structures that heart-dulness is difficult to detect at this level. The third interspace is skipped because the heart lies deeper there than lower down. The fourth interspace is selected and percussion started far to the right and carried along the

interspace to the left. The first slight diminution of resonance is noted. This may be due either to the heart or more often, I think, to narrowing of the interspace. Not far beyond the *right sternal margin* a point is found where resonance is moderately reduced and a definite sense of resistance is felt. This dulness is probably caused by the proximity of the heart, but may or may not correspond with the heart-border. Continuing toward the left, the right border of flatness or "superficial cardiac dulness" is reached. This point lies usually at the *left border of the sternum*.

When flatness extends across the lower end of the sternum or when moderate dulness extends more than half an inch to the right of the right sternal margin the heart is probably enlarged or displaced to the right. When dulness to the right at any of the points above mentioned seems excessive, another line of percussion is carried down the middle of the sternum from the manubrium to the ensiform. Along this line resonance normally diminishes slightly and progressively. An enlarged heart usually shows a decidedly more marked and more abrupt increase of dulness. Flatness across the lower end of the sternum at the level of the fourth interspace is a strong indication of enlargement.

The third step deals with the left border. After percussing from the top of the axilla to the base of the lung in the mid-axillary line, the fifth interspace is percussed, beginning at the mid-axilla. A slight diminution of resonance is encountered near the anterior axillary line or even farther out in many normal cases. Well-marked dulness in this region, however, is abnormal. Dulness suddenly increases as the mid-clavicular line is approached and flatness is found slightly further in, but flatness does not normally extend to the left of the mid-clavicular line, nor does well-marked dulness extend to the anterior axillary line unless the heart is enlarged or displaced or the chest of peculiar shape.

The normal limits of dulness as above stated have been determined by experience with the method described. Different standards of normal, similar in principle, might be set up by persons who percuss in a different way.

It will be seen that this standard of normal varies automatically with the size and shape of the chest. Thus it seems to adjust itself pretty satisfactorily to individual cases, because size and shape of

chest are known to be important factors in determining heart-size and shape or to bear a fairly constant relation to them.

When landmarks are used as here described, interpretation by body-weight or by size and shape of chest is required only under exceptional circumstances, such as unusual shape of chest or high or low position of diaphragm.

Inferences as to normality or the reverse of the heart, drawn from the percussion data, are stated in the form of opinions. For example, the cardiac dulness is described as slightly, moderately, or much increased to the right, to the left or in both directions. The reliability of the inference can be doubted, but the statement is worth face value as an opinion. Therefore it is not misleading.

Observation indicates that conclusions based on this or similar methods are as reliable, perhaps more reliable, than those based on interpreted measurements of percussion.

The inferences from percussion are checked in every case by palpation of the apex impulse with the patient sitting and again in the left lateral position, by auscultation, by enquiring for cardiac symptoms, and, when doubt remains, by estimation of blood-pressure and by radiological examination. By whatever method percussion is used, conclusions based on it should be checked in like manner.

Röntgenologists of experience judge of heart-size not by millimetres but by centimetres, because they recognize possible defects in technic and of interpretation which make it unsafe for them to form judgments to fractions of a centimetre. Percussion is a rougher method than röntgenology for determining heart-size or shape. Therefore, a margin of two centimetres should ordinarily be allowed for possible errors of technic or of interpretation when measurements or other data derived from percussion are advanced to indicate the presence or absence of enlargement. If this be granted, it becomes evident that increase of the transverse diameter by 2 cm. may exist without exceeding this margin of error, and, therefore, that such enlargement cannot be recognized with certainty by means of any method of percussion.

SOURCES OF ERROR AND SPECIAL VALUE

(a) Doctor Dodd first pointed out to me that there is a type of chest in which clinicians are apt to infer enlargement of the heart erro-

neously. It is the kind of chest which is box-like, relatively narrow at the base in front and flat on the sides. Cardiac dulness in these chests is greater than usual in the axillary region and the apex impulse may sometimes be felt unusually far out even though the X-ray fails to show any abnormality of the heart.

(b) The type of enlarged heart which percussion most often fails to demonstrate is that in which there is hypertrophy of the left ventricle with little or no dilatation. The actual measurements of such a heart are little changed; the apex impulse in the sitting position may give an indication of the condition, but when the patient lies on the left side the impulse will nearly always be felt farther out than a normal heart commonly swings, and it will be decidedly increased in force and width. Along with these changes may be found an accentuated aortic second sound and high blood-pressure. Symptoms or signs of failing compensation are not common at this stage of cardiac abnormality and tachycardia is absent. Measurements obtained by the X-ray may be normal or nearly so in these cases, but shape of the silhouette at the apex may indicate the true condition of the heart.

(c) While discussing cardiac measurements in an earlier paper, it was pointed out that a heart *originally small* might become enlarged¹¹ by several centimetres before any enlargement could be detected by measurements of the heart alone. Similarly, a long, narrow heart might become hypertrophied and yet give no demonstrable abnormality of dulness. The dulness ordinarily produced by the "drop" heart is well within the landmarks above mentioned and the superficial flat area may be absent.

(d) On the other hand, increased force of impulse, accentuation of sounds, tachycardia, and sometimes murmurs or temporary rise of blood-pressure may be encountered in cases of "irritable heart," but the cardiac impulse in the left lateral position will seldom be felt beyond the anterior axillary line, whereas hypertrophy usually carries it to, or nearly to, the mid-axilla. Cardiac dulness in these cases will not be increased but may be diminished because the group of "irritable hearts" includes a considerable proportion of "drop" hearts. For the recognition of "irritable heart" percussion seems to me to have a special value.

CONCLUSIONS

1. Recent radiological developments indicate that tables of measurements as standards of heart-size are likely to be supplanted by an index of relative heart-size obtained by comparing transverse diameter of the heart shadow with transverse internal diameter of the chest. If this index, after thorough trial, proves satisfactory, the problem of recognizing slight cardiac enlargement will be greatly simplified.

2. Owing to differences which are fundamental, radiological standards of heart-size are not satisfactorily comparable with measurements derived from percussion. The X-ray is very useful as a check upon inferences derived from the data afforded by percussion.

3. Measurements of cardiac dulness have not the mathematical accuracy which the figure implies. They represent little more than opinions, but the figures appear to have the significance of facts. Consequently they may be misleading.

4. Measurements made by one examiner cannot safely be used by another unless their methods of obtaining and of recording measurements are the same. It should be known, at any rate, how the measurements were obtained.

5. Measurements of dulness must be interpreted in relation to the chief factors which determine heart-shape and heart-size. Recorded measurements should, therefore, be accompanied by data as to body-weight and shape of chest and probable position of diaphragm. Further information is required in some cases.

6. A fairly constant ratio seems to exist between measurements of cardiac dulness and the circumference of the chest. If so, it might afford an index by which to judge of cardiac normality without reference to other facts.

7. While chaotic conditions relating to measurement of cardiac dulness prevail, and until some standard emerges which is worthy of general acceptance, the individual examiner seems most likely to get reliable results by adhering to the method in which he is most proficient.

8. Landmarks may be preferred to measurements.

9. A method of using percussion in relation to landmarks is described. Opinions so arrived at are stated as such. This kind of record has the advantage of simplicity and gives no fallacious appearance of mathematical accuracy.

10. Inferences as to heart-size or normality derived from percussion, by whatever method used, should be checked by other means.

11. A margin of 2 cm. should ordinarily be allowed for possible errors of technic or of interpretation when data derived from percussion are advanced to indicate the presence or absence of cardiac enlargement.

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RELAXED LUNG

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Definition.—A relaxed lung is one that is more relaxed or less tense than normal but still contains air. This relaxation results from the removal of some of the force required to maintain the lung in a state of normal expansion or distention. The reduction in this expansile force allows the lung to follow its natural tendency, which is to contract. This contraction allows the lung to become less distended, smaller, and altered in other ways. Atelectasis (literally, incomplete expansion) and collapse would be useful terms if they did not commonly signify total absence of air from the alveoli, that is, a foetal state of the lungs. Compression, as a descriptive word, implies an etiological factor not essential to pulmonary relaxation.

The relaxation may be slight in degree or marked. In distribution it may be diffuse throughout a considerable portion of the lung, as when adjacent to a fluid area in the pleura or pericardium, or it may occur as a disseminated process, small foci of relaxation alternating with foci of a different nature, such as emphysema and solidification, conditions encountered in broncho-pneumonias, tuberculosis, and irritation of the lungs from the inhalation of toxic gases. It is with the former relatively simple type and not with the latter much more complicated arrangement that this paper chiefly deals. The subject of atelectasis, that is, the foetal type of consolidation with approximation of the alveolar walls and disappearance of all air from the alveoli, will not be considered.

Importance.—The study of relaxed lung deserves attention for several reasons. Clinically the physical signs produced by relaxed lung may at one time offer diagnostic difficulties, and at another help to clarify the diagnosis of an obscure condition that is responsible for the relaxation. From the acoustic standpoint the study of relaxed lung has already yielded valuable information and promises further developments.

Clinical Manifestations.—Over a moderately relaxed lung underlying an area of several interspaces on the front of the chest, fairly characteristic, if not distinctive, signs are often revealed by percussion and auscultation. Percussion may furnish what Flint called an exaggerated vesiculo-tympanic note, with “the intensity greater than in health, the quality a compound of the vesicular and tympanic and the pitch more or less raised.” The quality and pitch are often decidedly abnormal but sometimes the increased intensity is the most striking feature. This note when localized, though occurring in a variety of different conditions, should suggest pulmonary relaxation in all doubtful cases.

Auscultation shows that the transmission of vibrations from the upper respiratory tract in typical cases of pulmonary relaxation is more marked than with normal lung, but these changes are more like those of partial or developing consolidation than those of complete consolidation. The vocal fremitus and vocal resonance are increased, but the changes from the normal in intensity are much more marked than those of quality or pitch. Marked degrees of bronchophony, as Flint has pointed out, are usually lacking. Whispered and breath sounds coming from the upper respiratory tract are not usually very loud and generally do not possess the high pitch or bronchial quality common over acute consolidation. Feeble breathing and weak broncho-vesicular breathing are often heard.

Diagnosis.—With typical signs over a fair-sized area of relaxed but otherwise normal lung the diagnosis is usually simple, if all the clinical data are assembled. A diagnosis of cavity has been made by competent men when the signs were due to relaxed lung and the signs may be more suggestive of cavity than many cavities provide. With a clear tympanic note on percussion, with increased vocal fremitus and resonance, with low-pitched broncho-vesicular breathing and whispered sounds, and with distinct râles—all these findings occurring at the upper part of the chest—one needs to be on his guard against calling an area of relaxed lung a cavity. Diagnostic aid may be obtained by observing a material change in the physical signs over the relaxed lung when the patient changes his position, due allowance being made for other conditions that may reveal postural variations in the signs. Again, the mistake has been made, as Austin Flint pointed out, of diagnosing a lesion on the normal side because the

percussion note is weaker than on the affected side and is interpreted as being impaired. Physical signs similar to those occurring over relaxed lung may be met with in cases of scattered infiltration, such as are found in some types of pneumonia and tuberculosis, and these signs may be encountered in the early stages of developing consolidation and in areas adjacent to consolidation. In these different conditions there may actually be foci of relaxed lung mingled with other foci of emphysema, infiltration, and congestion. A diffuse emphysematous process is not attended with the easily recognized increase in vocal fremitus and vocal resonance common to relaxed lung. The vesicular element of the breath sounds over the relaxed lung is usually feeble. A loud inspiratory vesicular sound over a relaxed lung can only occur if such a lung is functioning to a considerable extent. Thus a lung may expand and contract in response to respiratory movements even while continuing to be relaxed, and can absorb oxygen as long as it contains air. A lung outside the body, even though relaxed, will give a low-pitched modified vesicular murmur during the act of partial inflation. Typical signs of a relaxed lung over a localized area should in all doubtful cases make one suspect the possibility of some abnormal condition elsewhere, such as fluid. The findings elsewhere on the affected side may reveal the true condition where the relaxed lung exists. The X-ray may indicate the cause of the relaxation, and, in suitable cases, may furnish an area of increased density caused by the relaxed lung.

Clinical Cases.—To illustrate the physical signs of relaxed lung findings are here presented from a series of 18 cases of empyema seen in the empyema ward of U. S. Army General Hospital No. 14, Ft. Oglethorpe, Ga. This ward at the time was under the supervision of Lieutenant Colonel Martin, assisted by Captain Lee and Captain Furness. Twenty-seven other cases in the wards during the same period are omitted, most of these having been treated by intercostal drainage before I saw them. All of the 18 cases were males of draft age. The empyema occurred in 9 cases on the right side and in 9 on the left. Most of the cases developed after the epidemic of influenza prevalent in the fall of 1918 and the empyema in most of them was considered to be a complication of this disease. The duration period of the disease in the 18 cases from the onset of the first symptoms to the time I saw them varied from 17 to 75 days, the average

being 40.4 days. The symptoms of the different patients at the time of my observation varied in severity. All of the 18 progressed favorably. Pus was demonstrated in all of the 18 cases and all but two eventually had intercostal drainage, but the present records of physical signs antedate the insertion of the tube. The amount of fluid removed at any one time in the different cases varied from a few hundred cubic centimetres to about a litre, though this did not necessarily represent the total amount of fluid present in the pleural cavity at any one time.

The data referring to relaxed lung will be chiefly limited to the region above the definite signs of fluid. Only the front of the chest will be considered, the patients being generally in the recumbent position. Signs similar to those found anteriorly were at times found posteriorly but usually over a smaller area at the top of the chest. The long-continued recumbency favored the accumulation of fluid posterior as well as inferior (caudal) to the lung and this distribution of the fluid was frequently indicated by physical signs. The chest areas overlying the suspected relaxed lung included two or more interspaces and were not limited to narrow zones immediately adjacent to the fluid, these narrow zones, in fact, not usually presenting anything of special interest such as egophony. Marked differences on percussion and auscultation over relaxed lung were sometimes observed before and after the removal of fluid, the relaxed lung being still incompletely expanded after withdrawal of fluid.

The chest wall supposed to be in direct contact with the fluid itself showed, in most of the 18 cases, distinct diminution in vocal fremitus and resonance with marked dulness or flatness. Whispering pectoriloquy obtained in the same area was present in 10 of the 18 cases, was absent in 1, and was without record in 7 cases, probably being absent in most of these. The breath sounds through the fluid were feeble 11 times, bronchial 4 times, tubular once, absent once, and without record once. On the better functioning side, signs of compensatory emphysema were at times distinct, but at others the percussion note gave the impression of some impairment when compared to the louder note over the relaxed lung.

Inspection.—Anteriorly on the affected side 6 of the 18 cases showed some flattening, no record being made of the other 12 in this respect, though little if any bulging was encountered. In 2 of these

12 some fulness of the interspaces was noted. All of the 18 showed limitation of motion on the affected side—7 to a marked degree, 2 to a very slight degree. Seven showed better motion over the upper part of the affected side than the lower, and in 1 the motion was better below than above.

PHYSICAL SIGNS ABOVE DISTINCT FLUID LEVEL ANTERIORLY

No.	Side	Region	Percussion	V. F.	V. R.	Breathing	Whisper	Σ
1	R	3 rib up	Slight dulness	+	+	Diminished	?	1
2	R	Anterior	Hyperresonant	+	+	Weak Br. ves	?	2
3	R	3 rib up	Normal	+	+	Distant bronchial	Increased	3
4	R	4 rib up	Hyperresonant	+	+	?	Increased	4
5	R	3 rib up	Tymp. high pitch	N	+	Br. ves., low pitch	Incr. low pitch	5
6	R	4 rib up	Hyperresonant	+	+	Tubular	Increased	6
7	R	5 rib up	Hyperresonant	+	+	?	?	7
8	R	5 rib up	Normal	N	N	Normal	?	8
9	R	2-5 rib	Normal	?	+	Bronchial	Increased	9
10	L	4 rib up	Slight dulness	+	+	Insp. dimin., exp. prol.	Distant W.P.	10
11	L	4 rib up	Hyperresonant	+	+	Diminished	?	11
12	L	5 rib up	Tympany loud	-	N	Distant bronchial	?	12
13	L	5 rib up	Tympany very high pitch	N	N	Diminished	Absent	13
14	L	5 rib up	Hyperresonant	+	+	?	?	14
15	L	3 rib up	Tympany	-	N	Br. ves., low pitch	Faint	15
16	L	5 rib up	Tympany loud	N	N	Insp. dimin., exp. prol.	?	16
17	L	5 rib up	Hyperresonant	+	+	Normal	?	17
18	L	3 rib up	Normal	N	N	Normal	?	18

N., normal. +, increased. -, diminished. ?, no record. R., right. L., left. Up, extending at least to first rib.

In spite of the small number of cases, the long duration of many of them, and the lack of many interesting details, the general table of physical signs found over the upper part of the front of the chest on the affected side gives in a general way, except for a few of the cases, a fairly typical picture of the signs of relaxed lung. In 2 of the cases no abnormal signs were found above the fluid level in front, and in some of the others the diagnosis of relaxed lung is open to serious question.

The percussion notes above the fluid level anteriorly in the 18 cases give the following summary:

Normal 4 (Right 3, left 1)
Hyperresonant 7 (Right 4, left 3)
Tympanitic 5 (Right 1, left 4)
Slight dulness 2 (Right 1, left 1)

The term hyperresonance included one or more of these changes from the normal: intensification (never a subnormal intensity), elevation of pitch, or tympany, the latter with or without an associated vesicular quality.

The table shows in two-thirds of the cases a deviation from the normal in the direction of hyperresonance or tympany. It only gives an imperfect picture of the degree of the changes, but I am of the impression, that marked as these changes were in some of the cases, they were often less striking than in more recent fluid accumulations. The vesicular quality often persisted to some degree whatever other changes occurred.

Vocal fremitus and vocal resonance:

Vocal fremitus	18 cases
Normal	5 (Right 2, left 3)
Increased	10 (Right 6, left 4)
Diminished	2 (Right 0, left 2)
No record	1 (Right 1, left 0)
Vocal resonance	18 cases
Normal	6 (Right 1, left 5)
Increased	12 (Right 8, left 4)

These tables show an increase in the vocal fremitus and resonance which are common findings over relaxed lungs. Due allowance must be made for any failure to recognize the disparity in intensity normal to the two sides. The increase was not usually of a very marked grade, though at times it was very striking. The intensification was the most marked abnormal characteristic noted in the voice sounds. Some elevation of pitch was recorded in two of the cases, but the high pitch and bronchial quality of bronchophony, so conspicuous in many cases of acute consolidation, and even in some of the present series directly over the fluid area, played little part over the relaxed lung.

The breath sounds in eighteen cases:

Normal	3
Diminished	3
Diminished inspiration, prolonged expiration.....	2
Weak broncho-vesicular	1
Low pitched broncho-vesicular.....	2
Distant bronchial	2
Bronchial	1
Tubular	1
Not stated	3

The feebleness or absence of the vesicular inspiration in a large proportion of these cases is traceable to the small amount of air entering the alveoli in or near which the normal inspiratory vesicular murmur arises. Seven out of 12 cases with abnormal breath sounds show some bronchial character in the breath sounds, an indication, like the voice sounds, of better transmission of vibrations from the upper respiratory tract in the case of relaxed lung than that of normal lung. But in most of the cases with abnormal breath sounds this bronchial element is feeble or absent, and the respiratory sounds are usually not of the pure bronchial type commonly found over acute consolidation and sometimes over fluid.

Whisper.—The records about the whispered sounds are very deficient. Increased whispered sounds (not true whispered pectoriloquy) occurred in 5 cases, and a feeble whispered pectoriloquy in 2. About the 11 remaining cases there are no data as to the whisper, but probably the sound was absent in most of them. Clear high-pitched whispering pectoriloquy was more frequent directly over the fluid in these cases than over the relaxed lung. The records regarding the whisper, as far as they throw any light on the transmission of sounds from the upper respiratory tract through relaxed lung, are in keeping with the changes in the breath sounds.

Râles over the area of suspected relaxed lung were recorded in 5 of the 18 cases, but were not specially investigated after expiration and cough.

Case No. 2—Distant, moderate sized bubbling.

Case No. 6—Distant, mixed.

Case No. 10—Moderate sized bubbling.

Case No. 13—Obscure, moderate sized crackling.

Case No. 18—Few, indistinct.

Râles in cases like these occurring near the apex have distinct interest as suggesting possible tuberculosis. Their origin is not always clear, for they may be produced either in the relaxed lung or outside of it.

Summarizing the physical signs in the 18 cases we find in the majority of them, first, a percussion note that is louder or higher pitched or more tympanitic than normal, or combines two or all of these characteristics; secondly, an enfeeblement or disappearance of the normal vesicular inspiration; and, thirdly, a modification of the

sounds from the upper respiratory tract audible over the relaxed lung, as compared to the sounds over the normal lung. This change is one chiefly of intensification of the vocal vibrations, with comparatively little addition of the high pitch and bronchial quality characteristic of typical cases of acute consolidation. The picture suggests other conditions than relaxed lung, such as early developing consolidation, or disseminated foci of pneumonia or other infiltration of the lung. The amount of relaxation will modify the signs to some extent. Thus the tympanitic percussion note and the vocal resonance over relaxed lung may become decidedly lowered in pitch by the removal of fluid, the intensity of the sounds transmitted from the upper respiratory tract showing no change.

Any attempt to explain the differences in physical findings over normal and relaxed lung is hampered by our incomplete knowledge of the actual physical characteristics of a lung, either normally expanded or relaxed. The differences in tension in the tissues of the two kinds of lung are often so striking as to forcibly attract our attention to the subject of tension. It is well known that a healthy lung shows greater tension with inflation and less tension with deflation. Tension of the tissues in the lung recently removed from the body is much less than in its normal environment, but is not altogether lost, the lung ceasing to contract further because of the collapse of the small bronchi.

Percussion.—*Cæteris paribus*, the greater the tension of an elastic tissue or membrane the higher the pitch on percussion, but a normal lung often gives a lower pitched note than a relaxed lung. Evidently other complicating factors, of which many exist in the lung, require investigation. The relatively low pitch of the normal percussion note has been attributed (Norris and Landis, "Diseases of the Chest," 1918) to the pulmonary septa under normal tension acting as a load on the vibrations, tending to localize and delay them and thereby lower their pitch, just as wax attached to a tuning-fork reduces the number of vibrations, or as soapsuds placed in a bottle render the percussion pitch lower than when the bottle is empty. This load is reduced with the relaxation of the lung, which allows more of the lung to be set into vibration. With hypertrophic emphysema the pitch is likewise higher than normal and the reduced tension of the tissues in this case, as in relaxed lung, may also help to explain the higher pitch. A nor-

mal lung sufficiently expanded will give a higher pitched percussion note than when less distended. In the case of compensatory emphysema the degree of tension is not well understood, but the high-pitched note often found in this condition can hardly be traced to diminished tension as in the case of relaxed lung. Other marked physical differences may occur in compensatory emphysema and relaxed lung and yet both may give a percussion note that has much the same pitch, intensity, and quality. Relaxed lung rarely if ever decreases intensity, while emphysema may.

Transmission or Conduction of Sounds.—Some of the factors operative in the transmission of sounds through relaxed lung lend themselves to study with lungs removed from the body. The first successful comparative work on normal and relaxed lungs was done in 1884 in the classic experiment of Bullar (Bullar, J. F., *Proceedings of the Royal Society of London*, 1884, p. 411), who observed the different conducting qualities of a lung relaxed as compared to the same lung in a state of normal tension. Bullar constructed a chamber to resemble the thoracic cavity. In this he placed a collapsed lung with the trachea passing out of the chamber through a tightly-fitting opening. Another opening, also tightly fitting, admitted a stethoscope, the bell of which came in contact with the lung surface. A sort of bellows arrangement made it possible to exhaust the chamber of air, thereby effecting expansion of the lung. With such expansion of the lung the sounds admitted to the trachea became feebler, and with relaxation of the lung became louder. Bullar attributed the results to feeble conducting power of the distended lung but did not present any factors responsible for the enfeeblement. A much simpler, if less accurate experiment, can be conducted by introducing the lower limb of a Y-glass tube into the bronchus of a normal lung removed from the body, making the connection between the outside of the tube and bronchus airtight. One upper limb of the Y-tube is then connected with some source of sound like a tuning fork, the other limb being used to inflate the lung. As the lung is distended the sounds at the surface become weaker. It is easy to show that this de-intensification is not due simply to enlargement of the lung, because, distance for distance, the sounds pass through the relaxed lung with more intensity than through the distended lung. The enfeeblement of the transmitted sounds is due in considerable measure to the tension of

the pulmonary tissues. This can be illustrated in many simple ways; for example, by using a hollow tube which is cut in two pieces, a thin sheet of tissue of rubber or animal membrane being inserted between the divided ends, which are then replaced tightly so that the tube practically forms one piece as before, with a compartment to either side of the membrane. Sounds introduced into one end of the tube are audible at the other end, but the intensity decreases as the membrane is drawn more taut.

Summary.—Relaxed lung, as exemplified in a series of cases of empyema, may exist over an area of two or more interspaces. The physical signs are often characteristic if not distinctive. The percussion note is frequently changed in intensity (louder), in pitch (higher), and in quality (more tympanitic). If vesicular breathing is audible it is usually feeble. Through such a lung the vocal vibrations come with increased intensity but with relatively little change in pitch and quality from the normal.

The whispering and breathing sounds coming from the upper respiratory tract are frequently audible but usually weak. They are apt to be of a low-pitched tubular character and lack to a great degree the high pitch and peculiar bronchial quality frequently encountered over acute consolidation.

An important factor favoring the transmission of sounds from the upper respiratory tract through the relaxed, as compared to the normal lung, is the reduced tension of the delicate membranes of the pulmonary parenchyma. The relaxed tissues offer less impediment (reflection) to the vibrations than the tissues in a normal and more distended state.

I am very grateful to the surgical staff of U. S. Army General Hospital No. 14 for their coöperation in the work that made this paper possible, and I want particularly to thank Captain Blevins and Lieutenant Leib for valuable help in the medical care of the patients and in making clinical records.

AN UNUSUAL CASE OF ERYTHEMA MULTIFORME WITH HYPERLEUCOCYTOSIS, RIGORS, HYPERPYREXIA AND OTHER VISCERAL MANIFESTATIONS

FROM THE MEDICAL CLINIC OF THE PETER BENT BRIGHAM HOSPITAL, BOSTON, MASS.

By GEORGE R. HERRMANN, M.D.

HIGH leucocytosis, chills and high fever of a typically septic type are the most infrequent of the constitutional or visceral manifestations of the pure erythema group. Especially is this true of the cases coming within the circumscribed conception of the condition as defined by the earlier writers, Hebra¹ and Lewin.² The same also holds true in the broader groupings of Osler³ and Christian.⁴ Lewin² and also Parker⁵ report the occurrence of fever in a small percentage of cases, in which the temperature may be high, but is usually of short duration. Düring⁶ collected 105 cases, 31 of which showed moderate elevations of temperature. Case I in the series reported by Osler³ had fever, delirium and cold feet. No reports of chronic cases with chills and leucocytosis were found.

In a recent series of 47 cases of acute infectious origin, Guy⁷ reported, in most cases with extensive lesions, mild chills or chilly sensations, followed by irregular fever with evening rise and morning remissions, for two to three days in some cases and for fourteen to twenty-one days in others, ending by lysis. There was no instance of a temperature over 100°. The white blood cell count showed a slight polymorphonuclear leucocytosis which was rarely over 11,000. In thirty of these cases cultures from deep tonsillar crypts showed a hæmolytic streptococcus as the predominating organism. In nine cases a pyogenic streptococcus was found. In two cases in which the throat and tonsils were normal, a hæmolytic streptococcus was isolated from recent vaccination wounds. The serum of these patients did not agglutinate the predominating organism. After the erythema subsided, the hæmolytic streptococcus was not isolated, but when the exanthem recurred the *Streptococcus hæmolyticus* was again isolated. Other laboratory data was not remarkable. Only two of these

cases had had a previous attack, ten had recurrences a second time and one had a third recurrence.

Most of the other interesting and important visceral symptoms that have been reported by Osler, Christian and others were present in our case, as follows: There were prodromata for a day, a few days or even a week. The premonitory symptoms were usually vague, such as general malaise, headache, discomfort in the lower abdomen, cramps or stiffness in the legs (calves). The febrile erythema was initiated by pain in the lower abdomen, then gastro-intestinal crises with colic vomiting and diarrhoea. Occasionally there was enlargement of the liver, as demonstrated by palpitation. At times a rasping palpable friction rub was felt and heard over the front of the left chest, the patient has experienced this same sensation subjectively on previous occasions. There were aching pains in the joints and muscles. The fauces and pharynx were inflamed and congested, which fact corroborated the history of sore throats. The conjunctivitis was more marked on the left side. Small discrete glands were palpable in the epitrochlear, cervical, axillary and inguinal regions, thus presenting a general adenopathy.

CASE REPORT: The patient was first seen at the Children's Hospital in January, 1911. Since his first year of life he had been having peculiar attacks about once each year, in any season, but usually more severely in cold weather. The trouble would begin after a varying period of irritability and vague pains, with a chill lasting an hour, followed by a fever for about the same length of time. During the febrile period red blotches and segments of circles would appear all over the body. Great thirst accompanied the fever. At times the joints of the hands and feet were painful, this was associated with a "cold" and a sore throat, which were concomitant. The attack that brought him to the hospital in his eighth year had appeared at a shorter interval than a year from the previous one. Then, too, it was more severe, as it had already lasted two weeks, but the nightly emissions were becoming milder, indicating that the trouble was subsiding.

The physical examination was negative except for the blotchy erythema, an enlarged gland under the angle of the right mandible, a coated tongue and a soft systolic murmur heard just inside the apex of the heart. During his six days' stay in the hospital he had a

leucocytosis of 26,800 on one occasion and 37,300 on another. His urine was normal. The highest temperature recorded at this time was 99.2° , but it had only been taken bi-daily, morning and evening. Ward notes on successive days comment on the fact that his reaction and rash were most severe at about 4 A.M. It seems that his temperature must have been higher at this time.

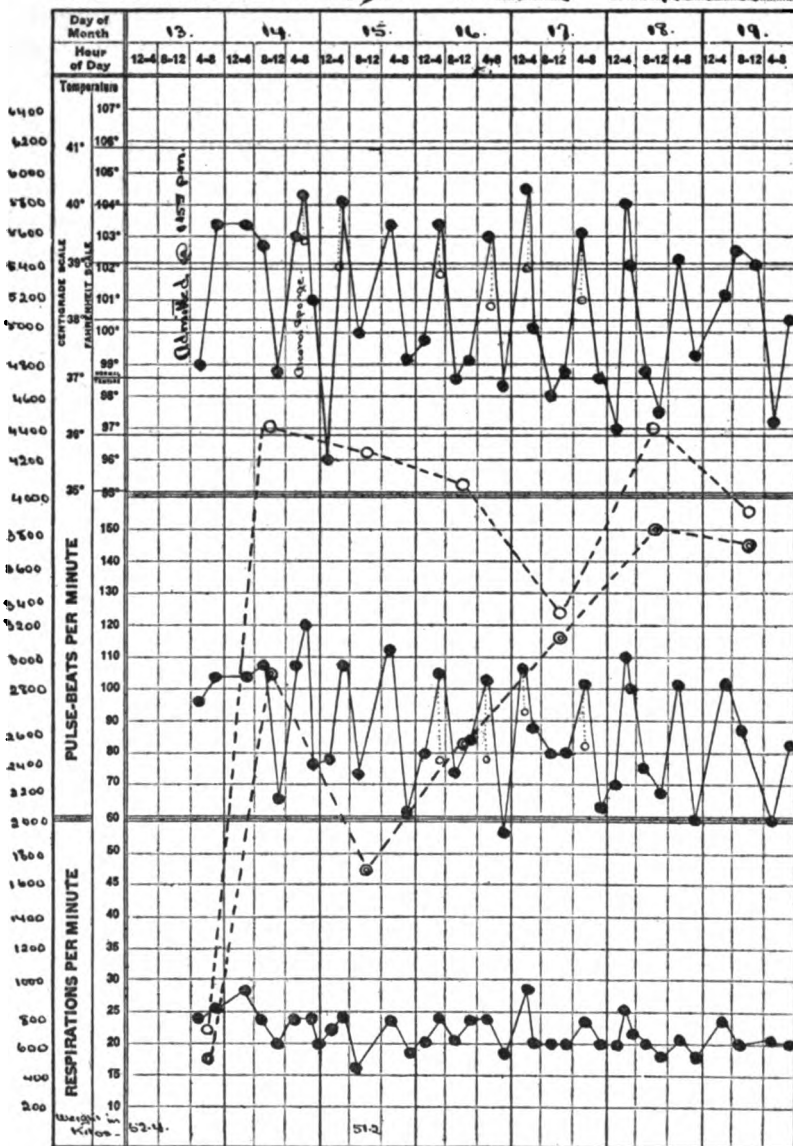
The patient was again seen at the Children's Hospital in a mild attack in March, 1911. He had been well until the week previously, when he had begun to feel feverish in the afternoons and to have an erythema of his arms and chest. As usual, a "cold" accompanied and followed the fever. The conjunctivæ had been injected since the onset of the attack. The bowels had been loose. The physical examination was the same as that on the first admission. The urine was normal. No blood counts were reported. During his five days' stay in the hospital, his highest recorded temperature was 101° on the evening of the first day and on the morning and evening of the second day. The next evening it was 99.6° and on the fourth evening it was 99.2° . At this point a four hourly chart was begun, but apparently the attack had waned. The highest reading was 100° at 4 A.M. and from that point there was a gradual, slightly interrupted drop to normal on the thirteenth day of the attack.

In August, 1911, he was seen in the out-patient department of the Children's Hospital. He had had a chill and febrile period for a week in July. Constipation, followed by diarrhœa and some joint pains had accompanied the attack. There was a tendency that the symptoms were most severe at night. His temperature was 99.6° at this visit.

In June, 1919, he entered the Peter Bent Brigham Hospital (Medical No. 11269) for the same trouble, which was now aggravated by accompanying severe gastro-intestinal symptoms. After leaving the Children's Hospital he had continued to have the affections at intervals of several months until about two years before admission, when they had begun to appear at shorter intervals. They were more severe as well as more frequent. He had just suffered a severe reaction period eight weeks previously. The present trouble had appeared a week before admission. As was the case in all his later attacks, he had had prodromal symptoms, such as discomfort or soreness in the lower abdomen, headache, malaise, constipation, followed by diarrhœa.

**PETER BENT BRIGHAM HOSPITAL
MEDICAL SERVICE**

NAME Walter P. Corcoran WARD 7-M DATE June 13, 1919.



In a few days the rash appeared followed by "hot spells" for an hour, without sweating, but with some itching of the skin, then came a severe chill, most often at night, this lasted about an hour, the rash then faded and the abdominal pain became slightly more intense. There was nausea followed by the vomiting of bile-stained material. His throat was sore and felt parched, consequently his thirst was intense and he would drink large amounts of water. The chills recurred every four to eight or twelve hours for one to two weeks. Throughout the period of the efflorescence he had a sore throat, blood-shot eyes, joint and muscular pains. He usually had vomited so much that now he would refrain from eating much during the attack, and consequently he lost from fifteen to twenty pounds in each one.

The past history is negative except for these attacks and whooping cough in 1911. He had been circumcised about thirteen years previously, and the tonsils and adenoids had been removed in 1909. Except as mentioned in the attacks he has had no eye, ear, nose or throat trouble. He suffered headaches only in the premonitory symptom complex. The cardio-respiratory history is negative except for a sense of shortness of breath or a feeling of inability to breathe deeply at times during the last few years.

Other than those mentioned, there have been no gastro-intestinal troubles.

He always had nocturia once, otherwise his genito-urinary history is normal.

The only relevant fact in his neuro-muscular history is that he had fainted twice in his attacks.

The physical examination showed a somewhat poorly nourished, tall boy of seventeen lying apparently comfortably in bed and cheerful in spite of the fact that he looked feverish and as though he had been very sick. Head is negative. Eyes show injected conjunctivæ, especially on the left. Ophthalmoscopic examination revealed normal fundi. The nasal septum is deflected to the left. The tongue is coated. The pharynx is injected or congested. A few small glands are palpable in the cervical chains.

There is an erythematous (macular, serpiginous, circinate and iris) rash over the whole trunk, especially on the back. The white line from pressure is quickly followed by a red line which persists for a few minutes. Thorax: Heart is negative except for a soft blowing

systolic murmur heard over the precordium. Blood pressure is 100 systolic and 65 diastolic. The lungs showed impaired resonance and harsh breathing at the right apex, but no râles. The abdomen is negative, but on one occasion the liver was palpable. The prostate gland is normal. Lymphatic glands are small and discrete, but definitely palpable in the neck, axillæ, groins and epitrochlear regions.

During his stay in the Peter Bent Brigham Hospital he continued to have two temperature rises between 103° and 104.4° every twenty-four hours for six days. The first rise was at about 4 A.M., and was always just preceded by a long hard chill, a real rigor of the same severity as a "malaria shake." The second rise came at about 4 P.M., and the accompanying chill was less constant and less severe. These facts are most strikingly shown in the accompanying charts. On the seventh and eighth days, there was only an afternoon rise to 100.5° . The patient had had the chills and fever for a week previous to admission, consequently this attack had lasted just two weeks. With the fever his fluid intake rose to four and one-half litres daily. Pain in the abdomen, nausea and vomiting followed the acute rises.

The leucocyte count rose to 42,200 at the height of the reaction. The high count persisted between 26,000 and 31,000 for a day after the temperature had remained normal, and then dropped daily down to between 9000 and 10,000 after the twelfth day in the hospital. The smear on the day of admission showed: polymorphonuclears, 86 per cent.; lymphocytes, 12 per cent.; large mononuclears, 2 per cent.; red-blood cells, normal; blood platelets, increased. The hæmoglobin was 85 per cent. No malarial parasites could be found, even after diligent searches with the concentrating technic of Bass and Johns.⁸ A blood smear on the day of discharge showed: polymorphonuclears, 56 per cent.; lymphocytes, 34 per cent.; large mononuclears, 4 per cent.; eosinophiles, 6 per cent.; mast cells, 1 per cent.; red-blood cells, normal. Blood platelets slightly increased. No blood parasites.

The admission specimen and two twenty-four-hour specimens of urine showed absolutely nothing abnormal, with the exception of the low specific gravity and color content, due to the physiologic diuresis.

The stools were negative for blood, pus, parasites and mucus.

Blood cultures taken at the time of a reaction were still negative after four days.

A fractional gastric analysis showed a perfectly normal curve.

NAME Walter P. Corsoran WARD 7m DATE June 20-1919



The chemical examinations of the vomitus were negative.

The blood serum Wassermann reaction was negative.

The phenol-sulphone phthalein test showed an excretion of 45 per cent., in 140 c.c. of urine after two hours and ten minutes.

The adrenalin test of Goetsch^{*} done by Miss Edna Tompkins after the patient's temperature had been normal one day gave a negative reaction.

Skin or cutaneous tests with the proteins of the commoner foods, animals, bacteria and pollens were done by Dr. I. Chandler Walker on the first day that the temperature was normal. All these tests were negative.

The report of the basal metabolism done by Miss Edna Tompkins was as follows: Height, 170 cm. Weight, 51.4 K. Buccal temperature, 97.6°. Blood-pressure, 100/50.

	Period 1	Period 2	Average
Pulse	51	51	51
Respiratory quotient	1.03	1.09	1.06
Volume per minute	7.42	8.17	7.80
Calories per hour	69.0	72.7	70.9
Metabolism (per cent. from normal)	Plus 1	Plus 7	Plus 4

Remarks: Patient somewhat restless in Period II. The quotient is unusual for a fasting patient. Patient positively denies any food since supper, and no mechanical cause can be given for such a value.

X-ray studies were carried out on the last day of the febrile period. The Röntgen Report: Plates of the sinuses show the right antrum to be slightly less radiant than the left. The Chest Plate: Diaphragm was apparently within normal limits. Hilus shadow showed increased density on both sides. Ascending branches of the bronchial tree show peribronchial thickening and beading. Markings seem to extend well up above the first rib, on both sides.

On the day on which he had the rasping palpable friction rub over the left front chest another X-ray plate was taken. The Röntgenologist reports as follows: Diaphragm is apparently within normal limits. There is a diffuse peribronchial thickening at the hilus along the course of the larger bronchi. Both upper chests above the third rib show an increased density, with peribronchial thickening and beading along the course of the bronchi. Left apex is less radiant than the right.

Among the therapeutic measures used, adrenalin 0.5 c.c. intra-

muscular at the height of the attack aggravated the subjective symptoms and increased the severity of the reaction. Atropine 0.001 gm. subcutaneously had no effect on the condition. Alkalies as sodium bicarbonate and calcium lactate had no beneficial or abortive effect on the course of the attack.

He was discharged well June 30, 1919, and told to return at the onset of the next attack. On September 1, 1919, he came to the outdoor department. He had been well except for constipation, which required saline cathartics every two days. Four days previously he had begun to feel weak, then his throat became sore and his eyes "blood-shot." There were also diarrhoea, tenderness of the abdomen, stiffness and soreness of the shoulders, stiffness and cramps in the legs and tenderness of the hamstring tendons and knee-joint on the right. The night before entering he began to feel thirsty and drank a few glasses of water. At about 2 o'clock on the afternoon of admission the thirst returned and he drank seven consecutive glasses of water. He had a "hot spell" and an erythema and perspired freely for about twenty minutes. He had the sensation of shortness of breath. In August, 1919, just one month previously, he had had a headache and an acute pain in the epigastrium for two or three days, but nothing further developed.

His physical examination was exactly as that on his first admission. He was sent into the hospital for further study.

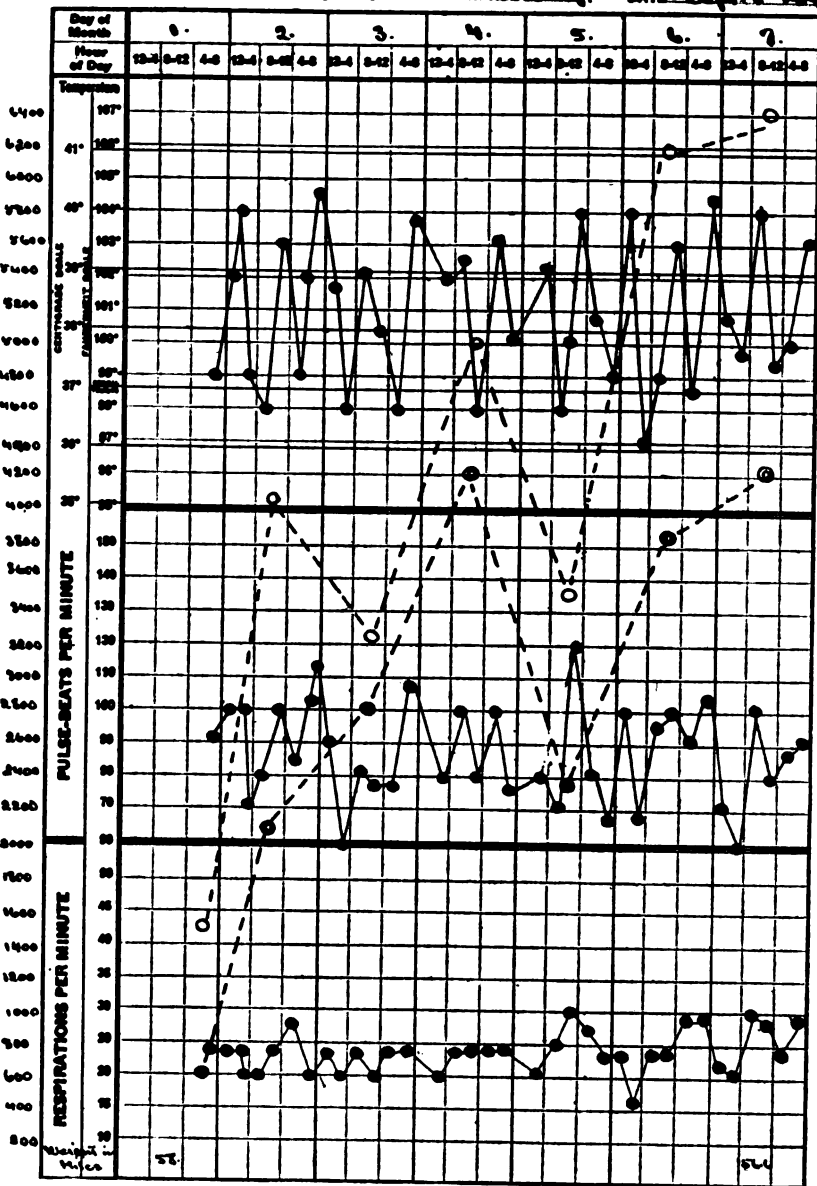
During his three weeks' stay in the hospital at this time, his condition ran about the same course as it had on his previous admission. His febrile period lasted for fifteen days and was slightly more irregular, with three high rises in twenty-four hours; namely, one after midnight, one at noon and one in the evening, rather than the two rises recorded in his first stay in the hospital. This is well shown in the accompanying four-hourly temperature charts. Chills were slightly irregular, but the severe rigors came at night as usual.

Daily leucocyte counts ranged between 20,000 and 35,000 during the febrile period and dropped to normal the day after the temperature became normal. The smear showed a differential count of 90 per cent. polymorphonuclears and 10 per cent. lymphocytes. The red-blood cells were normal. The hæmoglobin was 95 per cent.

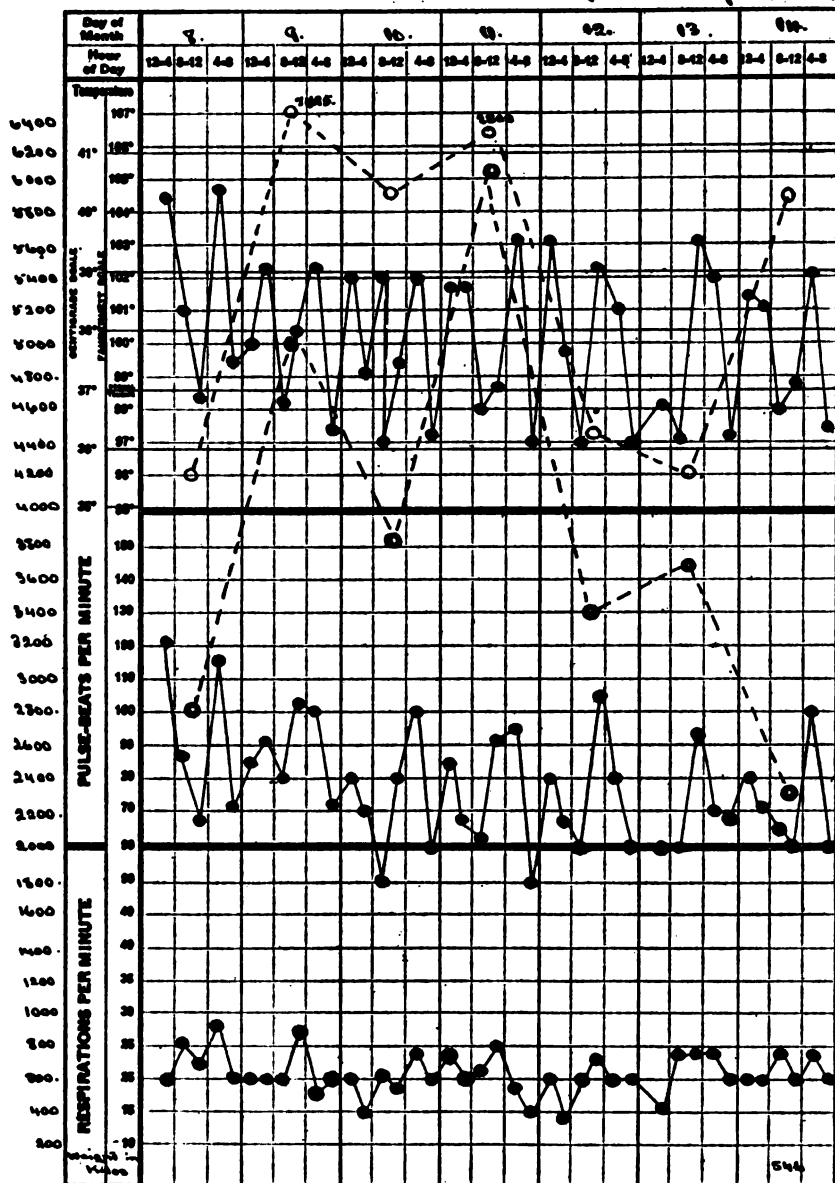
Urine analysis of the admission specimen and two twenty-four-hour specimens showed nothing abnormal.

PETER BENT BRIGHAM HOSPITAL
MEDICAL SERVICE

NAME Walter P. Corcoran, A. WARD 7th DATE Sept. 1st 1929

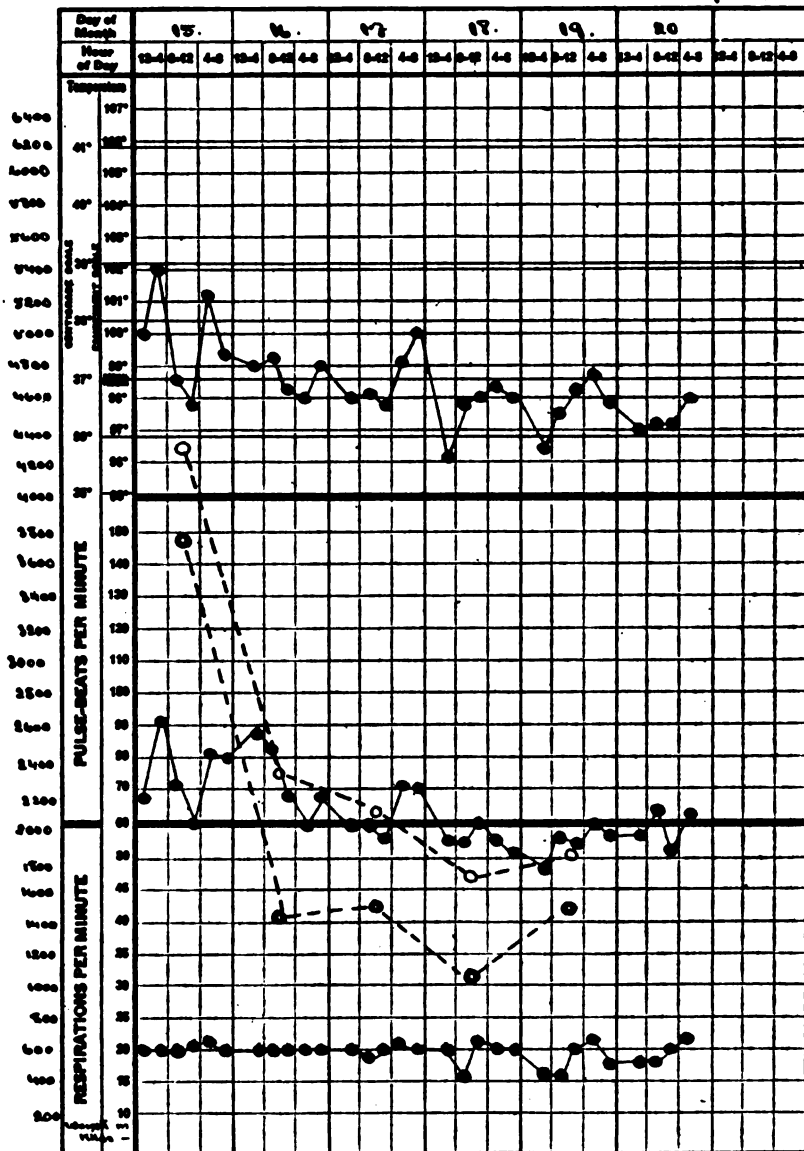


PETER BENT BRIGHAM HOSPITAL
MEDICAL SERVICE

NAME Walter P. ConwayWARD F.M.DATE Sept. 7, 1919.

PETER BENT BRIGHAM HOSPITAL
MEDICAL SERVICE

NAME Walter P. Cowdron WARD 31M DATE Sept. 15-1939



Blood cultures taken at the height of a reaction remained sterile. Stools and vomitus were negative for blood and abnormal constituents.

The blood-pressure at the peak of the temperature rise was systolic 100, diastolic 60. One c.c. of pituitrin was given intramuscularly and resulted in the disappearance of the erythema, but with no effect on the temperature, and the blood-pressure one-half hour after the injection was systolic 100, diastolic 48. The urine was kept alkaline for forty-eight hours with 90 gms. of sodium acetate without effect. A course of quinine sulphate was likewise of no avail in influencing or modifying the course of the condition.

Agglutination tests with cultures of colon bacillus, a gram positive bacillus and a gram positive chain forming streptococcus all isolated from the stools, as well as cultures of a staphylococcus isolated from the throat, were all negative.

The sinuses of the head, the jaws and teeth were again investigated by means of the X-ray. The Röntgenologist reports as follows: The right antrum is slightly less radiant than the left. Right ethmoids show slightly increased density. There is an area of decreased density about the apices of the right lower first molar. There is an area of increased density, probably an osteoma, at the apex of the root of the lower right central incisor. About the apex of the posterior root of the left lower first molar, there is also an area of increased density. About the root of the left upper bicuspid there is apparently a localized area of decreased density.

Barium studies of the gastro-intestinal canal by X-ray were made two days after the end of the febrile period. The X-ray report reads as follows: The stomach was normal in position, tone, outline with sluggish and irregular peristalsis. There was no gastric stasis. A good sphincter and first portion of the duodenum were seen. The ileum was not remarkable. At the end of six hours the head of the barium column had reached the cæcum and in twenty-four hours was in the rectum. The cæcum was normal in position and freely movable and contained a twenty-four-hour residue. The appendix was not seen. The colon was contracted in the transverse position, otherwise not remarkable.

In summing up the situation, we have a young man who all his life has suffered from increasingly frequent and severe periodic attacks of erythema multiforme, with unusual and severe constitutional symp-

toms and signs, and without a demonstrable cause or basis for the trouble. Erythema multiforme, with constitutional or visceral manifestations in initial or a few successive attacks within the span of a few years, as in the case reported by Guy⁷ and others, lend themselves to solution as results of infection, due to bacterial toxins directly or their by-products of metabolism, to foreign protein or pollen sensitization, to serum reaction, or drug idiosyncrasies. However, when the history, as in our case, extends over almost a score of years, beginning in the first year of life and gradually increasing in severity through adolescence, the problem is a difficult one, as is evident from our study.

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TWO CASES OF PRIMARY NEOPLASM OF THE CARDIAC VALVES

FROM THE PATHOLOGICAL INSTITUTE OF THE UNIVERSITY OF LAUSANNE,
SWITZERLAND

By F. C. FOREL, M.D.
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PRIMARY neoplasms of the endocardium are very rare, still more so are those of the cardiac valves. Therefore, the two following cases may not be devoid of interest.

Until now, only myxomata and fibromata have been described, and I shall record a type of new growth as yet not recorded in medical literature, but before so doing, the literature of the subject will be rapidly passed in review.

The oldest case on record is that of Curtis (*Archives de Physiologie Norm. et Pathologie*, iv, 1871-72). He found the following in a female subject, æt. eighty-three years. A small round tumor on the mitral valve, which was sclerosed, but offered no neoformed vessels nor any other sign of inflammation. Curtis did not regard this tumor as a true myxoma and considered that stroma of the neoplasm was composed of a tissue undergoing transformation, either tending to become fibrous tissue or derived therefrom. ✓

Debove (*Bull. de la Soc. Anat. de Paris*, 1873) found on the tricuspid of a female æt. fifty-four years, on its superior aspect, which was in no way otherwise pathologically changed, a growth the size of a cherry, bright red in color and soft in consistency. The neoplasm was attached to the valve by a rather short pedicle. Microscopical examination showed that it was composed of pure mucous tissue. No similar morbid change was found in any other of the viscera.

Guth (*Prager Med. Wochenschrift*, 1898, No. 8) described a freely pedunculated growth, the size of a pea, seated on the auricular aspect of the tricuspid. Some long villositities springing from a larger branch were covered by a perfectly regular endothelium. The stroma of the tumor, in general homogeneous, was in spots finely striated. A few rare capillaries were also noted.

Reitmann (*Zeitschr. f. Heilkunde*, xxvi, 1905) found a tumor about the size of a cherry on the anterior valve of the pulmonary, near Morgagni's nodule (in the semilunar valve). The growth was composed of villousities and formed by irregular fibrous tissue undergoing hyaline degeneration. Reitmann made a diagnosis of hyalo-fibroma and maintains that Guth is wrong in calling his tumor a myxoma.

Ribbert (*"Geschwülstlehre,"* 1904, p. 233) records three instances of valve tumors, which his assistant, Kesselring (*Thesis, Zurich*, 1900), describes as follows:

First case: An autopsy finding in an adult. Round tumor, located on the tricuspid one millimetre from its base. It measured three millimetres in height and in breadth, and was broadly inserted on the valve. Surface smooth, consistence soft, gelatinous, transparent and reddish in color. No specific stains were made to detect the presence of mucin, but the writer declares that the diagnosis of myxoma is fully justified by the microscopic examination.

Numerous vessels were noted in the subendothelial stratum of the valve, as well as in the growth itself. The vessels were of three kinds: arterioles and veinules, capillaries and full vascular buds undergoing development.

The writer also remarks that there were a few lymphocytes scattered through the tissue of the tumor, but not in sufficient number to justify an inflammatory origin. The stroma offered the structure of lax connective tissue, with round, oval or fusiform nuclei. The fundamental substance offered at times a fibrillar aspect, at others a perfectly homogeneous one.

The fibrillæ were especially marked at certain spots where the subendothelial stratum penetrated within the growth. Search for elastic fibres was not made.

The second of Ribbert's cases offered a uniform thickening over the entire surface of the tricuspid valve, which was twice its normal thickness. The principal growth was fissured, in height four millimetres, in breadth seven millimetres, and inserted on the valve by a thick pedicle. A second smaller tumor, placed beside the first, was somewhat confounded with it. There were no neoformed vessels in the valve.

The tissue composing the neoplasms was myxomatous in nature.

Both bipolar and multipolar cells were found, submerged in a homogeneous fundamental substance, or somewhat fibrillar. In the centre of the villositities, a kind of axis formed by connective tissue, could be seen, its origin being found at the base of the tumor and valve. The entire surface of the neoplasm was covered by an endothelial layer, the cells of which were very rich in protoplasm.

The third case of Ribbert showed a thickened valve, with a mushroom-like growth having numerous villositities. The tumor measured 2.5 mm. in breadth and 1.5 mm. in height. The pedicle was one millimetre in diameter.

Numerous villositities, similar to the neoplasm in structure, projected from the valve itself, alongside the tumor. The growth was composed of very distinct endothelium and by a tissue rather more fibrous than myxomatous, but here and there hyalin degeneration was met with.

In Ribbert's fourth case the tumor was 1.5 x 1 millimetre, located on the pulmonary valve, which itself was slightly thickened. There were also some aberrant villositities arising from the valve itself beside the neoplasm. In the slightly striated stroma, round or elongated nuclei could be seen. The endothelial cells were much more elongated and regular than in the three preceding cases.

In the second, third and fourth cases of Ribbert, no vessels could be found, a fact to be noted.

Leonhardt (*Virchow's Archiv.*, B. 181, 1905) found in a tuberculous girl, æt. twenty-two years, a mushroom-like tumor developing from the auricular aspect of the posterior mitral valve. The valve itself presented all the stigmata of chronic endocarditis. The tumor measured 5 x 6 millimetres and was located very near the insertion of the valve, being attached to it by a short, broad pedicle. In color the growth was reddish-brown, its surface smooth and free from all fibrinous deposit. In consistency it was soft.

Microscopically the valve was found thickened from endocarditis. The neoplasm was composed by a mass of lax connective tissue, poor in cells but with numerous vessels filled with erythrocytes and leucocytes, communicating by way of the pedicle with the neoformed vessels which were scattered over the valve. The tumor tissue was composed by a homogeneous mass, which the fixing fluid had rendered finely granular and fibrillar in certain spots and which offered the various

reactions of mucin (thionin, muci-carmin). In this tissue fusiform and stellate cells were found, and which being united by their anastomoses, formed a fine network.

Extravasated erythrocytes and leucocytes were seen in the stroma, likewise a large quantity of brownish pigment. The connective tissue which accompanied the vessels spread over the surface of the neoplasm like a capsule, and on this capsule there was an endothelial layer continuous with that of the endocardium. On staining, it was made evident that the elastic fibres of the tumor accompanied the blood-vessels and only the vascularized portions of the growth presented them. Leonhardt concludes that his case was a very vascularized myxoma, his diagnosis being based upon very distinct specific stains.

To Djewitzki (*Virchow's Archiv.*, B. 185, 1906) we owe the first case of tumor of the aortic valve. It was a myxoma. At the autopsy of a male, æt. thirty-eight years, he found a neoplasm inserted upon an otherwise normal valve. It was mammillated, the size of a cherry-stone and situated on the ventricular aspect of the valve, a little to the left of Morgagni's nodule. It was pedunculated and its surface presented numerous papillæ, which gave the growth a velvety look; some of the papillæ were given off on the pedicle itself.

The writer noted elongated cells with a clear nucleus, with little protoplasm and presenting prolongations or long connective filaments with a fusiform nucleus, these being situated at the periphery of the neoplasm.

The filaments of connective tissue, staining red with Van Gieson, ran to the centre of each papilla. The elastic fibres were arranged in a network at the base of the growth and intermingled with the connective-tissue fibres. In the valve the subendothelial stratum was very thick at the level of the pedicle, while elsewhere it was hardly visible. The other strata composing the valve had not undergone any morbid change. At no point was there the slightest trace of vascular neoformation.

I have briefly summarized these ten cases which, in spite of the considerable similarities they possessed, nevertheless differ from each other in some detail. The first case of my own, which I shall now describe quite closely, resembles Guth's case and Ribbert's third case, but I have not been able to find a single case reported in the literature which in any way resembles my second case.

CASE I.—Marie U., æt. sixty years, came to autopsy with a diagnosis of hepatic cirrhosis. A small, transparent, gelatinous tumor was found inserted on the posterior aortic valve. This neoplasm measured $6 \times 6 \times 4$ millimetres, was on the free border of the valve five millimetres to the left of Morgagni's nodule. It was composed of small transparent and soft villosities, which gave the surface of the growth a granular look. The growth was not pedunculated and was inserted on the free edge of the valve by a large base. The other aortic valves, as well as the pulmonary, tricuspid and mitral, were absolutely normal.

The ventricles were slightly dilated and hypertrophied, but at no spot was the parietal endocardium pathologically changed.

Serial sections of the tumor and valve on which it was implanted were made and stained according to the usual methods. (Hemalum combined with eosin or with Van Gieson, thionin, mucicarmin, and lastly lugol and methyl violet with sulphuric acid.)

The valve was normal in its membranous portion and no vessel could be detected. At the point where the little tumor emerged the free border of the valve was thickened, and from this thickening a formation similar to a tree, whose trunk inclined on the free border of the valve, the branches being represented by the ramified villosities, arose. (See Fig. 1.)

The tumor was almost everywhere with a very variable endothelium, excepting on the thickened portion of the valve on its side looking towards the aorta. On the ventricular aspect the cells were cubic, almost cylindrical in shape. Their protoplasm was clear. On the villosities they were flattened, occasionally desquamating. The stroma of the principal trunk of the neoplasm was composed of fibrous tissue with elongated nuclei, striated and staining red with Van Gieson's fluid.

However, certain portions were stained in light rose and this was particularly the case of the trunk of the growth. In certain spots elongated nuclei and a fibrous structure could be found, but at others the section presented a perfectly homogenous aspect. As to the villosities, they had almost the characteristics of connective tissue at times, at others they were already in a state of degeneration, but still capable of staining a pale rose hue. Others there were presenting a perfectly homogeneous aspect and took no stain whatever.

It is curious to note that there where the mass which formed the centre of the villosity a granular aspect was offered and arranged in a kind of ring or crown, like the hooklets of a tenia in its vesicle.

Staining with thionin or mucicarmin did not reveal the slightest trace of mucous tissue anywhere in the neoplasm.

Staining with lugol and sulphuric acid did not reveal an evidence of amyloid degeneration.

This case macroscopically is very like those of Guth, Reitmann, Djewitzki, and the second, third and fourth cases of Ribbert. But

FIG. 1.



Tumor of aortic valve.

the microscopical examination of the tumor showed that those portions evidently composed of fibrous tissue which stained red with Van Gieson, were distinctly striated and with elongated nuclei, so that I could only conclude in favor of a diagnosis of fibroma.

But this fibroma had undergone changes, but not amyloid or mucous degeneration, as the staining amply proved. The greater part of the neoplasm and the majority of its villosities presented a homogeneous appearance; no fibrillar network, striation, cell or nucleus could be seen. They took on a uniformly gray tint with every staining method. Occasionally this substance was granular. There-

fore, we were dealing with a hyalin degeneration, probably caused by poor nutrition from the absolute absence of any vessels whatsoever. Consequently the only logical diagnosis is that of a fibroma with hyalin degeneration.

Among the valvular growths mentioned in the foregoing pages, there are some which have no resemblance to the small neoplasm I have described (cases of Curtis, Debove and Leonhardt). On the contrary, others seem to belong to the same class of new growths.

Djewitzki, who very conscientiously examined his specimen and submitted it to specific reactions, has clearly placed mucin in evidence in the fundamental tissue of his tumor. It is, therefore, unquestionable that certain gelatinous and papilliferous formations may be authentic examples of myxomata.

Ribbert's third case is perhaps a neoplasm similar to mine. It was likewise a villous growth of soft consistency and having a gelatinous aspect. Kesselring declares that all these neoplasms are myxomata; nevertheless, as I have pointed out, the fact is not proved beyond a doubt.

Unquestionably there exist fibromata inserted on the cardiac valves. Curtis stated at the time he wrote that his specimen was composed of tissue in transition, either working towards a fibrous state or returning therefrom. Reitmann speaks of hyalofibroma, and in my case just reported the diagnosis of fibroma with hyalin degeneration does not appear to be subject to objections.

As my second case differs in every particular with those that I have been able to collect in the literature, I shall take the liberty of giving a rather detailed account:

CASE II.—Bertha C., æt. thirty-six years. Pernicious post-partum anæmia; 15 per cent. hæmoglobin. A small amount of serous transudate in abdominal cavity, the pleura and pericardium and pulmonary oedema. Spleen enlarged, red and firm. Profound anæmia of all the viscera, but without manifest morbid changes.

The heart is small, limp and brownish in color. On the auricular aspect of the posterior mitral valve and exactly at its middle, is a small discoid excrescence with a smooth surface and soft consistency, measuring about 2.5 millimetres in diameter. It is gray in color. The growth lies on the valvular endocardium and attached to it by a pedicle (see Fig. 2) 270 μ in thickness. The neoplasm is not covered by a

thrombus. There is no other valvular change of the mitral nor of the other cardiac valves. The parietal endocardium is perfectly normal.

This was fixed in sublimate, and after paraffin inclusion serial sections were made and stained according to the usual methods, such as hemalum or ferric hematoxylin and eosin or Van Gieson, and finally with Weigert's methylene blue, in order to study the network of elastic fibres.

The valve upon which the neoplasm is seated is 840 μ in thickness, perfectly regular in outline and offers no other morbid change. In sections comprising both the valve and tumor, the following strata are encountered:

(1) The endothelium (Fig. 2, end) of the valvular aspect is composed of large cells with a flat nucleus; (2) a layer of connective-tissue cells, which the section has cut perpendicularly; (3) a layer of longitudinal muscular fibres; that is to say, extending from the point of implantation of the valve to its free border; (4) a very thick layer of connective tissue, with fibres going in the same direction, and which forms the most considerable and resistant portion of the valve; (5) a thin layer of transversal connective-tissue fibres; (6) a very delicate reticulated membrane containing a few sparse nuclei and serving as substratum for the endothelium; (7) the endothelium of the ventricular aspect of the valve.

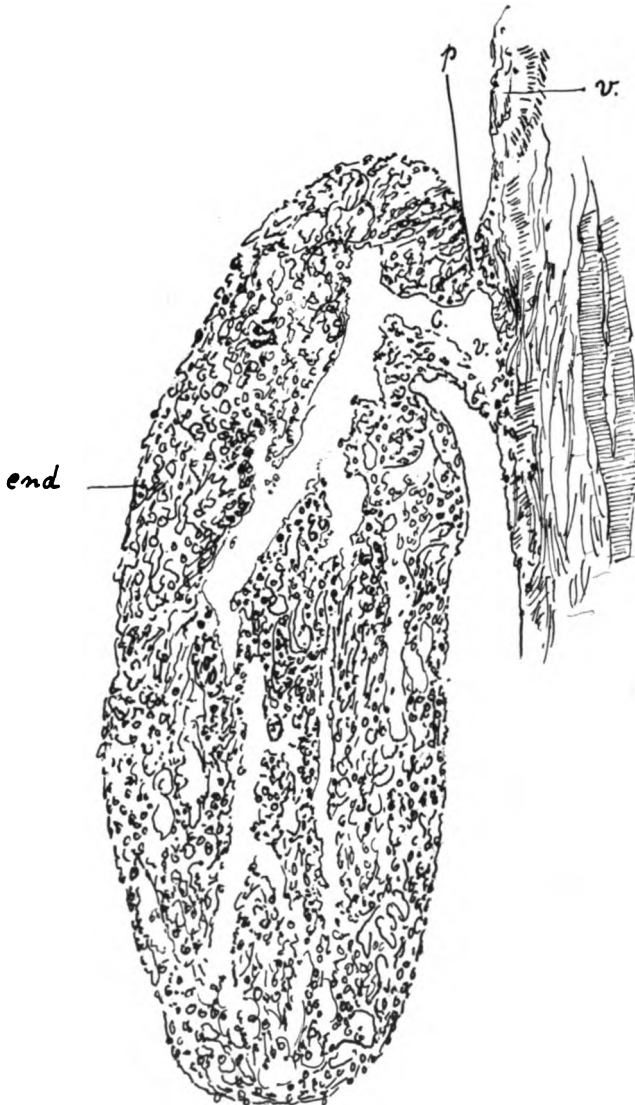
In the subendothelial layer, small, round or flattened vessels, lined with endothelium are seen (Fig. 2, v).

In the direct neighborhood of the growth layer No. 1 penetrates into layer No. 2. In point of fact, it will be seen that some endothelial cells leave the surface and take the direction of the depths of the growth. By becoming mixed with the connective tissue of the subendothelial layer they give rise to a mixed tissue, which in every way resembles the stroma of the neoplasm and its pedicle.

There is no appreciable limit between the endothelial and subendothelial layers and the stroma of the growth.

Examined at a low power, a section passing through the tumor shows that the growth has the shape of a flattened mushroom whose stalk takes birth from the valve. This pedicle is covered with endothelium continuing directly from that of the valvular endocardium,

FIG. 2.



Histology of tumor from mitral valve (see text for description).

but which becomes irregular, cubic in the first place, then almost cylindrical. Its cells are pressed against each other.

The stroma of the pedicle is composed of a network of connective-tissue fibres, which contain large cells with a clear protoplasm in its meshes. The protoplasm is slightly granular and the nucleus a little elongated.

In the very middle of the pedicle is a canal (Fig. 2, c) lined with endothelium. It ends in the growth in a number of ramifications, and at its other extremity continues in the valve by a kind of vessel similar to those already mentioned, containing no blood and only a few desquamated cells.

As I have said, the canal ramifies in the neoplasm, first giving off two main ramifications, and these give rise to ramuscles in number, which are met with in the sections cut longitudinally or transversely, but whose calibre does not diminish sensibly and never reaches the size of a true capillary.

This lacunar network (see Fig. 3, Frontispiece) is not everywhere lined with endothelium. This is detached in certain spots, while in other areas the tumor tissue proper offers some rather irregular hiati, which resemble lymphatic fissures.

The stroma through which these canals run is composed of rather fine connective tissue. In the meshes of this tissue large, oval or polygonal cells of various sizes are lodged (Fig. 3, end, Frontispiece). They measure from $8 \times 12 \mu$ to $7 \times 12 \mu$. Their nucleus, which is round or oval, is situated in the middle of the cell. They stain well and measure $4 \times 6 \mu$. Each cell is isolated in its alveola and invariably separated from other cells by a connective-tissue fibril or two.

In the lumen of the lacunar system numerous cells of varying size and types are seen.

(1) Cells with much protoplasm (Fig. 3, end, Frontispiece), oval in shape, measuring $8 \times 12 \mu$, with an equally ovoid nucleus, with its axis directed in the axis of the cell, and which measures $5 \times 6 \mu$. These cells are quite numerous, and some of them are adherent to the walls of the vessels. Their protoplasm is granular, their nuclei stain very differently and in quite a number of them there are one or several vacuolæ.

(2) Cells with a large nucleus, which measure from $7 \times 5 \mu$ to $9 \times 6 \mu$. Their protoplasm is very thin and hardly visible. It melts

into numerous droplets, which form a circle around the nucleus, giving it a denticulated look. As to the nucleus itself, it took the stain poorly in most cells; occasionally it stained well with hemalum.

(3) Small cells, few in number, measuring $3 \times 3 \mu$, staining well and in every respect recalling lymphocytes.

Lastly, here and there are some polynuclear cells, measuring $13 \times 15 \mu$. There are no plasmatic cells.

Weigert's stain does not reveal any elastic fibres in the neoplasm.

We are here dealing with a true neoplasm. The valve offers a perfectly normal structure and its thickness is everywhere the same. The connective-tissue fibres do not offer the irregular arrangement that is found in valves the site of an endocarditis, neither are any irregular blood-vessels to be seen, penetrating in depth; there is no fibrous nor red-blood corpuscles in the stroma of the neoplasm, neither is there any pigment.

Consequently, we certainly were not dealing either with an organized thrombus or an endocarditic vegetation, but with a genuine neoplasm.

Now, what is its nature? It is composed of canals having an arborescent arrangement, separated from each other by a very thin connective-tissue stroma, containing large numerous cells with a granular protoplasm (Fig. 3, end, Frontispiece).

Do these cells characterize the neoplasm or should more importance be attributed to the canals which run through it? In this case the intercanalicular connective tissue and endothelium would be of secondary importance.

From whence do the cells come which line the canals, likewise the cell elements met with between the canals in the meshes of the connective tissue? It seems to me probable that these cells are derived from a proliferation of the valvular endothelium, with which they directly continue, without any definite limits and with which they have a certain resemblance. The diagnosis of this case is, therefore, an endothelioma of the mitral valve.

Should one regard the vessels especially as representing the characteristic portions of the growth, it might be labelled angioma or even lymphangioma, and, in point of fact, I shall endeavor to show that these canals are lymphatic in nature.

Here are the reasons that we offer to corroborate our point of

view: (a) The structure of these canals which, in the neoplasm, are only composed of endothelium and show no elastic fibrillæ. (b) They do not contain a single red-blood corpuscle. (c) They terminate in cul-de-sacs and also in fissures similar to lymphatic lacunæ. (d) In the pedicle there is but a single vessel; the walls are so thin that at the most it could be a vein, and were this so, there is no afferent artery present.

Moreover, only various types of leucocytes are encountered, especially mononuclears (the polynuclears are very rare) and large cells that I am inclined to classify under the heading of endothelial cells.

If the reader will turn to the description given, he will perceive that these large clear cells have a granular protoplasm and one or several vacuolæ which never encroach upon the nucleus.

The cells lining the canals are of the same size and shape and present the same granular protoplasm as well as the same phenomena of vacuolization.

Finally, be it noted that all degrees are to be found from the cells adherent to the walls, those commencing to become detached and those floating freely in the lumen of the canal.

Did the patient's general condition have any influence on the nutrition of the neoplastic cells? It is, in fact, possible that there was a relation between cause and effect, between the pernicious anæmia and the fact that the epithelial cells became detached from the walls, therefore, they were degenerated.

On which aspect of the valve are valvular growths generally situated? It is curious to note that those in all the cases reported where precise data on this point are given, the neoplasm was seated on the aspect of the valve in contact with the blood, or should it be preferred, on that which locks with the other homonym valves.

Because their localization corresponds with that of endocarditic vegetations is not a sufficient reason to invoke inflammation as necessary in the genesis of the two instances of neoplasm I have reported, but an indefinitely repeated traumatism cannot be eliminated from the etiology.

What is the importance of the vascularization of the valve in the differential diagnosis between neoplasm and endocarditis? I do not think it is very great. Several perfectly authentic neoplasms were found to be very vascular, as well as the valve supporting them.

Others, quite as characteristic, were deprived of any vascular supply, likewise the valve bearing the growth.

According to my way of thinking, vascularization is one of the signs by which an endocarditic process can be recognized; but this is, of course, a very insufficient character upon which alone to base a diagnosis of inflammation.

The neoplasms which I have passed in review can consequently be classified, according to my personal views, based on the description given by their reporters, as follows:

Myxomata, cases of Debove, Guth, Leonhardt, Djewitzki and Ribbert's case one and two.

Fibromata, cases of Curtis, Reitmann, Ribbert's third and fourth case and my first case.

Endothelioma, my second case.

To sum up, it can be said that the general primary neoplasms of the cardiac valves exist; that they are very uncommon, small and usually of no import so far as clinical symptoms are concerned; that they are indifferently situated on all the cardiac valves, they may or may not be vascularized; and, lastly, that up to date three types have been found, viz.: fibroma, myxoma and endothelioma.

TUBERCULOSIS AND THE RED CROSS

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TUBERCULOSIS is the disease having the greatest number of victims. It attacks 20 per cent. of humanity and kills from one-sixth to one-seventh part of the world's population. It is propagated by contagion, and the agent of contagion is Koch's bacillus, discovered in 1882.

This bacterium may remain for a long lapse of time enclosed within a lymph-node, in a bone or even the lung; this is the so-called *closed* tuberculosis, and is not contagious. But as soon as the lung ulcerates and suppurates, the bacilli are expelled with the sputum and then we have an *open* tuberculosis, which can be transmitted to those living with the patient by the fine droplets that he scatters when in the act of coughing, or indirectly by the dust formed by the dried sputum on handkerchiefs, floors or objects which the patient has brought in contact with his mouth.

It has been demonstrated that the virulence of the bacillus may persist in darkness for months, or even years; that, on the contrary, solar light destroys it in a few hours, from whence arises the danger of habitations deprived of sunlight. Thus it forms foci of infection in unhealthy dwellings, into which the sun never enters, and which are overcrowded and dirty, not to say filthy.

There is, therefore, in the struggle against tuberculosis as a social peril a part to be fulfilled by the municipal authorities, by the State, which should establish the sanitary status of houses and adopt measures of prophylaxis, by proper sanitation of dwellings and workshops.

Paris has given us an example to be imitated. The sanitary condition of all the houses of the capital was established by the work of M. Juillerat, and showed that the evolution of tuberculosis is unquestionably the outcome of insalubrious housing and overcrowding. The

death-rate of tuberculosis is in direct ratio with the number of stories and in inverse ratio with the dimension of the court-yards and inside air-shafts. It also becomes evident from these investigations that out of a total of 81,500 houses within the limits of Paris, 5000 were tuberculosis *nidi*, distributed in areas whose contours were marked out on the plans of the city. Among these fateful dwellings, furnished lodging houses were distinguished by the highest mortality from this disease; it attained 20 per 1000 inhabitants, while it has an average of 5 per 1000 in the populous districts of Paris.

It is calculated that there are 12,000 deaths annually from tuberculosis in Paris. In order to estimate the Parisian contagion, about an equal number of provincial immigrants, who return to their own homes to die, should be added. These poor creatures infect their respective villages, thus creating foci of tuberculosis in the country and even into the valleys of the Alps.

If human contagion is indubitably the most important source of the spread of tuberculosis, one should nevertheless not overlook—as Koch was inclined to—milk derived from tuberculous cows, whose consumption, when unboiled, is the undoubted origin of tuberculation of the intestine, peritoneum and mesenteric lymph-nodes in children.*

The experiments and clinical observations of Bang, of Copenhagen; Demme, of Berne, and Gosse, of Geneva, have proved it.

To remedy this disquieting extension of tuberculosis among cattle, it is essential to adopt strict means of inspection and supervision, such as have been employed in Denmark, Norway and Sweden for a number of years, to the great benefit of the public health. This most important part of the struggle against tuberculosis escapes action on the part of the Red Cross, as it belongs to the domain of the State.

In England, since the year 1836, legislation intervened to favor associations and companies which built working people's houses. From 1868 to 1882 a series of laws were promulgated (Dwelling Improvements Acts) which obliged municipalities to demolish unhealthy

* D'Espine. Report on Infantile Tuberculosis, Paris Congress, 1900. *Idem*. Report on Infantile Tuberculosis, Congress for the Protection of Childhood, Brussels, 1907.

dwellings and to furnish an abode to those who, by these measures, were left without asylum.

In the space of fifteen years, England spent something like £120,000,000, a portion of which was used for the destruction of unhealthy quarters and houses, another portion for measures of disinfection wisely enacted; and, what is still more important, they were rigorously carried out. By this sanitary legislation Great Britain, more than any other European country, lowered its mortality from tuberculosis. While in 1842, 38 per 10,000 subjects died annually from the disease, in 1900 the death-rate had dropped to 13 per 10,000.

Besides government action, the most active agent, one with a far greater field of usefulness, is unquestionably an *anti-tuberculosis education* of the people, with a view to creating "*sanitary morals*," according to Professor Landouzey's apt expression, without which the best sanitary laws, misunderstood and imperfectly applied, remain a dead letter.

This education should be given, in the first place, to the patient himself, impressing upon him the danger of his sputum.

Next, the mothers should be instructed on all points necessary for preserving their children from contagion. Later on, school-children should receive instruction, likewise young girls attending high school, where it seems to me very useful, because by so doing we can initiate the future mothers in the dangers of tuberculosis which lie in wait at the conjugal hearth and for the child from the cradle on.

As I showed in my report on "Tuberculosis and the School," at the International Congress at Rome in 1912, the teachers in the infantile primary schools should inculcate in their young charges practical anti-tuberculosis hygiene, the interdiction to expectorate on the ground, the dangers of kissing, the importance of hand washing before each meal and the ills ensuing from the biting of the nails, since onychophagic children become, more than others, tuberculous (Derecq).

Anti-tuberculosis instruction for adults is of inestimable usefulness. It can be accomplished by printed leaves, by pictures, and by ambulant museums of tuberculosis, which go from town to town, as is done in Switzerland. This is a most important branch of the anti-tuberculosis activity of the Red Cross.

We have only to refer to the admirable campaign undertaken in

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France by the American Red Cross, which went through all the Departments in automobiles, distributing illustrated pamphlets everywhere, destined to engrave upon the simplest mind how tuberculosis is contracted and in what way it can be avoided.

After having explained the prophylaxis, we should next examine those means which have been recognized as the most apt for the struggle against the disease *per se*. In the first place, is tuberculosis curable? At the time that I began my medical studies at Paris, in 1864, the prognosis of pulmonary tuberculosis was more than dark. The great French phthisiologist Louis, who had been my father's teacher, often talked to me of the futility of treatment of pulmonary tuberculosis. At this epoch all that was prescribed in hospital practice was cod-liver oil and calcium phosphate.

Since the discovery of the specific bacillus, a specific medication, similar to that discovered by Yersin and Roux for diphtheria, has been thought to have been found. But unfortunately up to the date of writing neither Koch's tuberculin nor the various anti-tuberculosis sera put upon the market have been able to convince the majority of the medical corps of their specificity.

The disease is the expression of the struggle between the bacillus and the human organism. It is by fortifying the organism by hygienic measures that modern medicine has been able to cure a large number of tuberculosis cases, especially when the disease has been taken in hand at its onset.

For that matter, the curability of tuberculosis is a fact well known in the past, and even Hippocrates says: "Phthisis, if treated from the onset, can be cured."

This spontaneous curability is proved by the frequency of healed tuberculous lesions discovered at autopsy. Brouardel found evidences of healed tuberculosis in 50 per cent. of autopsies performed on subjects whose death had been accidental. This fact demonstrates the importance of the soil upon which the seeds of tuberculosis fall.

Among the most important arms at our disposal for the struggle against tuberculosis, I would mention two; namely, the *anti-tuberculosis dispensary* on the one hand, and the *aerium* and the *sanatorium*.

The first anti-tuberculosis dispensary was founded at Lille by Professor Calmette; it has been working since November, 1901, and has served as a model for hundreds of similar institutions in France and

Belgium in the first place, then in Germany, Switzerland, England and the United States. The dispensary has become the corner-stone of the struggle against this disease.

Its activity consists in hunting out tuberculosis by sending investigators to houses, by teaching prophylaxis and carrying out sanitation in the homes of the poor.

The dispensary physicians do not limit their services to consultations; they sort out the cases, sending those who are the least infected to free air cures or to popular *sanatoria*. They hospitalize advanced cases of phthisis who have become permanent dangers for the family.

The rôle of women as house visitors among the tuberculous has been brought into relief by the remarkable report read by Mlle. Chaptel at the Rome Congress. It amply demonstrates what an important part is reserved for the Red Cross and its corps of nurses as an auxiliary to the work carried on by the anti-tuberculosis dispensaries.

The preventive action of the dispensaries could also be greatly increased if the Red Cross should place its sanitary units at their disposal for the successive disinfection of dwellings of tuberculous subjects, who so frequently change their abode.

The *æriums* and *sanatoriums*, which render such immense service in the cure of tuberculosis, are the practical demonstration of the ancient proverb: "*Aer, pabulum vitæ.*"

The *ærium* or *sanatorium* of fortune, according to the picturesque expression of our colleague, Professor Brunon, of Rouen, is a gallery constructed for the open-air cure, where the patients remain lying down during the day and are properly fed. These *æriums* should be constructed in all urban populations, or at least in their immediate neighborhood.

To attain the same end, open-air schools have been constructed at Geneva, Lausanne and elsewhere, for weak children or those suspected of having tuberculosis.

The sanatorium is an institution for the treatment of all curable cases of tuberculosis, and has been rendered accessible to the poor in almost all countries by the creation of free institutions.

Germany, which at present possesses more than eighty sanatoriums, has made the free sanatorium the principal machinery of a complete preventive and curative system, which has been given over to

the Offices of Insurance Against Disease, Invalidism and Old Age, to which for the past thirty years the law has compelled both employers and employees to belong. The application of this law having revealed that the principal cause of invalidism was tuberculosis, the insurance offices opened up sanatoria.

Switzerland has shown the importance of altitude for the cure of tuberculosis by founding sanatoria at an altitude of more than one thousand metres at Davos, Arosa, Leysin, Montana, etc. We possess to-day twelve free sanatoria which have received, from 1910 to 1914, 11,149 tuberculous patients, 56.9 per cent. of whom have been cured; that is to say, have been able to return to their ordinary vocations.

The superiority of altitude sanatoria is made clearly evident by the ultimate results of cure. Seventy-nine per cent. of the patients treated at the Bâle Free Sanatorium at Davos had maintained their full working capacity after the lapse of three years, and 58 per cent. at the end of six years.*

Beside the altitude treatment of pulmonary tuberculosis, the brilliant results obtained in the cure of surgical tuberculosis by means of the solar rays combined with altitude at Samaden by Doctor Bernhard, and at Leysin by Doctor Rollier, should be mentioned.

The maritime sanatoria play a part similar to those of altitude, but they are more particularly suitable for that form of tuberculosis known by the name of scrofula. In my report to the Paris Congress in 1905, on maritime sanatoria, I endeavored to show that I considered the sea-side cure as superior to that of altitude in certain forms of lymph-node or bone tuberculosis.

France alone has now more than twenty sanatoria built along her sea-coasts and a total of 4000 beds for sea-air cure.

The anti-tuberculosis struggle, inaugurated in France in 1886 by Professor Verneuil, has taken on a world-wide development with the advent of the twentieth century and comprises powerful associates in nearly every country. The International Anti-tuberculosis Association, founded at Berlin in 1902, was for a long time presided over by Professor Brouardel, and after his death by Mr. Léon Bourgeois, ex-Minister of France and an untiring promoter of social hygiene.

The International Association founded annual meetings, the ninth

* Ganguillet. *La Lutte antituberculeuse en Suisse*, 1917, page 338.

and last having taken place at Berlin in 1913, as well as the International Congress, which holds its meetings every three or four years (Paris, 1905; Washington, 1908; Rome, 1912). Thanks to the mobilization of all the social forces—both public and private—official or voluntary—the efficiency of the struggle entered into has been clearly demonstrated by a general and gradual diminution of the death-rate from tuberculosis in nearly all countries.

Unfortunately, during the four years of war which the world has traversed, tuberculosis has raised its head everywhere. The Allied prisoners in Germany and Austria have been attacked in fearful proportions, due to insufficient food and overwork and drudgery far above their physical capacity. The danger of dissemination of tuberculosis by the return of soldiers to their homes is only too evident, and it is here that the Red Cross can and should enter upon the scene.

The question of tuberculosis in the army has preoccupied hygienists for some years past. Our colleague, Dr. F. Ferrière, presented a report at the meeting of the Red Cross Societies held at London in 1907, on the intervention of the Red Cross in favor of men who had been rejected at recruitment, as well as soldiers eliminated from the army for the reason that they were tuberculous or suspected that they might develop the disease.

One of the Societies of the French Red Cross—Union des Femmes de France—had decided some years ago to participate in the anti-tuberculosis fight by looking after soldiers upon their return to their families and seeing to it that they were not lost sight of. A general plan of the work includes the creation at several places in France, as well as in Algeria and Tunisia, of open-air establishments, where young soldiers, suspected of having tuberculosis or slightly infected with the disease, can be employed in agricultural work. To-day this effort should be intensified by the Societies of the Red Cross throughout the world.

As a conclusion to this very concise and necessarily incomplete summary, I believe that at present an active coöperation imposes itself upon the Societies of the Red Cross and the anti-tuberculosis organizations now existing in all countries.

Such assistance could be realized by a participation in the anti-tuberculosis education of the masses, by the utilization of the sanitary personnel in the work of the dispensaries and of disinfection, by the

creation of sanatoriums and by the use of the wooden buildings belonging to the Red Cross for open-air cures, and, finally, by the creation of agricultural colonies, where the cure of tuberculous soldiers can be assured by a return to moderate physical activity in hygienic conditions which are wanting in his home.

NOTE.—As a word of warning, it is to be pointed out to the American profession that in *Prof. D'Espine's paper the establishments of Dr. Schnöller and Dr. Dannegger at Davos are not to be considered, for obvious reasons.* (Translator's note.)

Obstetrics

ABDOMINAL PREGNANCY WITH LIVING FŒTUS

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EVEN the most casual study of the subject, or a few months association with an abdominal surgeon enjoying a reasonably large practice, will convince the most skeptical that impregnation of the ovum outside the uterine cavity with extra-uterine development of the fœtus, is by no means uncommon. It must also be remembered that the number of cases in which accurate diagnosis is made is much smaller than the total number of patients applying for treatment, and that the latter by no means represents the actual number having ectopic gestation. In many instances the patient remains for weeks or months under the care of the general practitioner, being treated for ovaritis, pelvic hæmatocele, pelvic abscess, peritonitis, appendicitis, salpingitis, etc.

A review of the literature of the last decade induces the conviction that the surgeon is in large measure to be blamed because the general practitioner does not possess greater knowledge concerning ectopic gestation than he now has. It must be obvious that, for the most part, all definite pathological knowledge has come to us from the operating table and the mortuary slab. No one has the opportunity for investigating pathology in the living subject so frequently as has the surgeon. To him is granted the privilege of actually seeing and handling the diseased tissues, the possession of which is causing the patient pain and distress.

The writer believes that had abdominal surgeons talked and written as much about ectopic gestation during the last twenty years, as they have about appendicitis and gall-bladder disease, the profession and even the public would have greatly benefited thereby. The women who are the subjects of ectopic gestation quite naturally know absolutely nothing about their condition, and many of the general practitioners who first see these patients possess only a meagre idea

of the abnormal occurrence, its frequency, symptomatology, the indications for surgical intervention, etc. The majority of them appear to believe that ectopic gestation is exceedingly rare, and therefore they are never on the lookout for it. While others admit its relative frequency, they do not devote the necessary time to consideration of the symptoms to insure accuracy in diagnosis. A rather large proportion have not had sufficient experience in the physical examination of women to be accurate in their interpretation of the clinical findings when their investigation is completed.

Why does pregnancy occur outside the uterus, when in all other respects the efforts of nature are directed toward production of the natural? This is a difficult question to answer with any degree of certainty. After a careful study of 177 cases of ectopic gestation, Mall became convinced that in every instance there had been an inflammatory lesion involving the Fallopian tube before lodgement of the ovum therein; in fact, anything interfering with the ciliated epithelium and a continuous stream of fluid from the ovary to the uterus—or even the slowing of this stream—would tend to favor tubal pregnancy. The history of nearly every case seems to be in favor of this theory.

The most frequent site for the ovum to become attached to the tube is in the ampulla. This is due to enlargement of the tube at that point and to its vascularity. The fertilized ovum soon buries itself in the wall of the tube. It does this by means of the tropoblast. All the tissues are affected—muscular, connective tissue, blood-vessels. There is, however, no well-formed decidua as in uterine pregnancy, and from a practical standpoint the damage done the blood-vessels is the most important. The walls are eroded by the tropoblast, just as occurs in the uterine vessels in the formation of the inter-villous space. Sooner or later the tendency is for the ovum to have its connection with the tubal walls disturbed by reason of minute intramural hemorrhages.

With the ovum embedded in the ampulla several terminations are possible:

- (a) Tubal abortion, complete or incomplete.
- (b) Tubal rupture.
- (c) Formation of a mole, with subsequent changes—atrophy or disintegration.
- (d) Continuance of the pregnancy to the later months or even term.

If tubal abortion occurs the tube soon resumes its normal appearance, just as the uterus does after expelling its contents prematurely. Everything then "quiets down," hæmatocele forms, and in time disappears.

When rupture occurs as a rule it is between the sixth and eighth week, although it may occur much earlier. The rupture and accompanying hemorrhage may take place into the general peritoneal cavity or between the layers of the broad ligaments, as the tubes are not completely surrounded by peritoneum, being uncovered along the lower part of the wall, where the layers of the broad ligament come together.

Rupture directly into the peritoneal cavity is much the most frequent termination, as might be expected, and is more serious, for there is no let nor hindrance to the effused blood. When the whole ovum is suddenly expelled into the peritoneal cavity, it usually dies. It is questionable if it can re-ingraft itself on a peritoneal surface. Sometimes, however, where the pregnancy has advanced farther and rupture is gradual, the placenta remains attached in the tube and develops there and in the surrounding tissues, while the ovum grows in the free abdominal cavity. I believe the specimen here illustrated is one in which the ovum was expelled from the tube and which lived and engrafted itself to the surrounding structures.

It is possible for pregnancy to advance to full term without rupture of the sac; and in the opinion of the writer the case here shown would have been one of that kind, as the sac at the time of removal was very thin but still intact. The sac was adherent to the surrounding structures which displaced them to a greater or less extent.

The second most frequent place for the ovum to become attached is in the isthmus of the tube. Here rupture is peculiarly frequent and occurs generally at a very early date, often, indeed, as early as the second or third week and before any menstrual period is missed. The explanation of this early rupture is that the muscular fibres are peculiarly scanty and poorly developed, so that the ovum readily perforates the tubal wall.

The next most frequent form is in the infundibulum of the tube or the elongated ovarian fimbriæ, the occurrence of which is very rare. The ovum becomes either separated or contracting adhesions to the surrounding parts continue to develop.

One of the rarer forms of extra-uterine pregnancy is that in which the ovum is implanted in the interstitial portion of the tube. Werth in 120 cases did not find a single instance of this nature. In this form of extra-uterine pregnancy the uterus is found much enlarged in one cornu. In a typical form of this variety the ovum, which is attached between the uterine and abdominal openings, grows on the wall of the uterus and "dissects up" the muscular layers. The cornu of the uterus is pushed upward, the sac enlarging especially in that direction. Sooner or later rupture occurs, occasionally into the uterine cavity, but most commonly into the peritoneal cavity. It is attended by very profuse hemorrhage, as large vessels are usually torn. It is generally stated that rupture is a late occurrence, often not taking place until the fifteenth or sixteenth week, but this is questioned by a table given a number of years ago by Werth which showed that this rupture was not so frequent as one time supposed.

Everyone acknowledges the possibilities of an ovarian pregnancy. There are several well authenticated cases which have been described within the last few years. Rupture has occurred at a comparatively early date. An especially interesting case is one described by Menge—an ovarian pregnancy of nearly full term, with a coexisting uterine pregnancy. The diagnosis of tumor obstructing labor was made. When the tumor was opened it was discovered to contain a fully-developed fœtus.

It is a well-known clinical fact that in the majority of cases of tubal gestation, the patient has previously given birth to one or more children, then remained sterile for several years. This would indicate the presence of tubal pathology which prevented impregnation for a considerable period of time, and if gestation had not occurred the tube would have eventually become normal or nearly so.

The reason impregnation does not occur in a badly inflamed tube, is that the ovum rapidly disintegrates, and marked changes occur in the tubal lumen. The epithelial folds become hypertrophied and the tips adhere, forming cavities. Again, the epithelium may "out-pocket" into the muscular wall, and in this way obstruct the lumen. While the ovum may be grasped by the fimbriæ and started in its progress toward the uterus, either of the abnormalities mentioned may so retard its descent that it becomes too large to traverse the tubal lumen, and lodges in the outer end if the mucosa is normal

there, or it may lodge near the uterus if the lumen is constricted at that point.

While all authors admit the frequency of pregnancy outside the uterus, the abdominal variety developing outside the tubal lumen is less common, and especially is this true where living children are removed from the abdomen, as in the case to be herein reported.

From a careful study of the subject, it is believed that two types of abdominal pregnancy may occur: First, the ovum may become impregnated near the outer extremity of the tube, and by contraction of the muscular wall the product of conception is forced backward into the abdomen, and there finding attachment further growth is favored. Second, primary abdominal pregnancy, which is questioned by many authors, but at the present time the trend of opinion is that this variety can and does occur at times in the human being. Bell (*Surg. Gynec. and Obstet.*, 1911) reports the case of a primary abdominal pregnancy in a rabbit, impregnation occurring during confinement of the animal in the laboratory where its abdominal cavity had been inspected on two different occasions during the prosecution of experimental work. He concluded that as primary abdominal gestation occurred in animals, it might happen just as readily in the human being.

Believing that a résumé of the symptomatology and diagnosis of ectopic gestation might not be amiss, the writer has reviewed the literature for the last few years, not only of abdominal pregnancy with living children, but also tubal gestation with and without rupture. Such a review is interesting, not only because of its bearing upon the question of diagnosis, but also the morbidity and mortality of patients seeking relief.

No one can deny that the earlier accurate diagnosis is made and surgical treatment instituted, the lower will be the maternal mortality. While perhaps few of the patients die from hemorrhage, *per se*, yet a certain number of them do; many others succumb to infection of the blood-clot which accumulates within the abdominal cavity; a still smaller number perish as a result of adhesions between the intestinal folds and obstruction to the fæcal outflow thus produced.

Ectopic gestation is also interesting because of the tendency to recurrence in the same individual. Stuart McGuire (1913) tabulated 2998 cases where the patients were operated upon for tubal

pregnancy, with 113 recurrences, or 3.8 per cent. He did not believe these figures were accurate as to the number of recurrences, as a careful record could not be kept of the post-operative history of all the patients applying to the various surgeons for relief.

Study of these cases indicates that vascularity at the placental site is extensive even at the third month, that it is markedly increased from the third to the sixth month, and that the larger percentage of deaths which occur in attempting to relieve these patients may be attributed to hemorrhage; next from infection occurring in the placenta where it is left in the abdominal cavity, or in the placental site when it is removed. This, of course, does not take into consideration other dangers incident to enucleation of the placenta from its abnormal site.

A careful study of the symptomatology of ectopic gestation should enable the physician to make an early accurate diagnosis, and thereby obviate the dangers which have been enumerated. The symptoms present in the average case will be about as follows: The majority of the patients are multiparæ, and, having already experienced one or more pregnancies, believe they understand their condition. In most cases there is a history of sterility for several years, and which has persisted until they noticed symptoms which they interpreted as another pregnancy. About one-third of the patients will have nausea, vomiting, more or less mammary disturbance, and the general systemic discomfort noted in practically all cases of normal gestation.

Upon being questioned as to their menstrual history, nearly all of them will state that they missed one period, that uterine hemorrhage occurred about the time the next period was expected, and accompanying this there was noted the passage per vaginam of more or less *débris*, which they interpreted as an early miscarriage. This uterine flow, as a rule, continues much longer than normal menstruation. During the period of amenorrhœa little or no pelvic pain is experienced, possibly not one-third of the patients giving a history of distress which they cannot readily ascribe to other causes. Physical examination reveals the uterus enlarged and more or less softened, and the color and consistency of the cervix frequently changed. If the uterine discharge be subjected to washing and careful examination shreds of decidua will usually be found.

Prior to the time when hemorrhage begins, the slight distress felt

in the pelvis is due to the growing ovum, hence is not marked. After hemorrhage occurs the symptoms are caused by traumatic or irritative lesions, rupture of the sac, peritoneal irritation from extravasation of blood, etc.

If the temperature be taken it will be found these patients have from one to three and a half degrees of fever. This, of course, applies after the primary lowering of the temperature, due to excessive hemorrhage, in cases where this has occurred. It has been the writer's experience that in very few instances is hemorrhage so marked as to cause the typical shock and collapse described in textbooks. Most patients have a slow hemorrhage, not sufficient at any time to produce symptoms of shock.

The character of the pain usually conforms to certain rules, *viz.*, it is in the lower portion of the abdomen, it is for the most part confined to the affected side, rarely is it referred to the entire abdominal cavity. In most cases the pain is described as "cramp-like or colicky," in a few where the symptoms are typical it is stated to be "knife-like." Pain is periodic and more or less paroxysmal. It is periodic, because tubal rupture is associated with repeated small hemorrhages which distend its lumen, the leakage of blood irritating the peritoneal surfaces and producing intense pain similar to that observed in abscess formation. It is paroxysmal, because of the character of the tube, the peritoneal irritation producing a condition simulating intestinal peristalsis.

In cases where, after a sharp "stab-pain," the typical shock and collapse appear, the symptoms are due partly to hemorrhage and partly to irritation of the peritoneum by the extravasated blood. Rarely do we see typical pain and collapse repeated in the same case. Where marked hemorrhage occurs, of course the pulse-rate is increased in direct proportion to the amount of blood lost. In milder degrees of hemorrhage, the pulse-rate is only slightly increased, the range being from 85 to 100.

Symptoms referable to the enteric tract are present in many cases. Irritation of the peritoneum in the region of the pelvic colon gives the patient a desire to frequently defecate, and the act is accompanied by more or less straining due to the sensation of incomplete evacuation. Frequency of urination is also a common manifestation.

Examination of the abdomen before rupture and extravasation of

blood will reveal little information of value. Vaginal examination carefully made discloses tenderness on the affected side with enlargement of the tube and a more or less "doughy" feel. After rupture and the formation of adhesions, the uterus will be found partially fixed, the *cul-de-sac* bulging, and tenderness on the affected side. At this time abdominal examination will show more or less tympany with increased tenderness upon pressure, and if the gestation has progressed far enough, a mass may be felt occupying the affected side, or even the entire pelvic cavity. This enlargement is due to adhesions between the intestinal folds.

When the foregoing clinical symptoms are present, little difficulty should be experienced in arriving at the conclusion that ectopic gestation has occurred.

On April 23, 1916, I was requested by Doctor Layman, of Cecilia, Kentucky, to see Mrs. S., white, aged thirty-three years. The patient was five feet six inches in height, of rather slender build, and a housekeeper by occupation. The family history was good; both parents died of pneumonia; one sister died of typhoid fever two years ago. Personal history: Menstruation was established at thirteen, had always been regular, but she suffered more or less pain during each period. She was married at twenty-seven, became pregnant within three months, and gave birth to a perfectly healthy child one year after marriage. Two years later she again became pregnant, and a second healthy child was born in due time. There is no complications during either pregnancy or accouchement. Both children are living and healthy. The husband gives no history of specific disease.

Present illness: In February, 1916, the patient began having more or less pain and distress in the left pelvic region. Little attention was given this at the time, as her mother was dangerously ill and had been for several weeks, and finally died during that month. The patient remembered having menstruated in January, but could not recall whether or not the February period appeared. The anxiety during this time, together with the worry and grief caused by the death of her mother, rendered this part of the history uncertain. She did recall, however, that she missed the period in March, nor did she menstruate at what would have been her regular time in April.

Six or seven weeks prior to my examination she recalled that,

one morning when arising from the bed and assuming the upright position, she was seized with severe pain in her left side, which produced some nausea and compelled her to again retire. On the succeeding morning, while attempting to dress and prepare breakfast, another seizure of pain occurred, which again compelled her to return to bed. There was no history of collapse, such as is oftentimes observed in ectopic gestation. From that time she was compelled to remain in bed most of the time, due to pain in her left side, which, as she expressed it, was so severe that it precluded her standing on her feet longer than a few minutes at a time.

Four or five weeks prior to my examination, Doctor Layman had detected by bimanual examination a mass in the pelvis which he thought was the enlarged uterus, and since the patient had been having a slight uterine flow for two or three days, he diagnosed pregnancy with threatened abortion. The mass grew rapidly in size, and the flow ceased in three or four days. This caused a change in his opinion, and after further observation a tentative diagnosis of ectopic pregnancy was made.

During my examination the uterus was easily outlined, being separated from the main tumor-mass by a distinct sulcus. The abdominal tumor at this time filled the entire left side below the umbilicus, and extended nearly across the right side. This mass was irregular in outline, dull on percussion, extremely tender upon bimanual palpation, and the uterus was firmly fixed in the pelvis.

There was no history of any recent uterine flow, there were no mammary changes, no nausea nor vomiting; in other words, the usual symptoms of pregnancy were absent. The patient's temperature each day varied from $99\frac{1}{2}^{\circ}$ to 102° F. The pulse-rate on slight exertion would increase to 100, the patient showed marked emaciation, and anæmia was pronounced. There was considerable pain during defecation and micturition.

With the foregoing history, which was obtained during an imperfect examination in a farm house, the diagnosis was made of ectopic gestation which had probably become "walled off," permitting only sufficient loss of blood from time to time to continue the anæmia. The patient was informed accordingly, and, together with her husband, the matter was thoroughly discussed, both as to the risk of allowing her to remain longer at home, and the dangers of post-

poning abdominal section longer than a few days. After due consideration of the facts, they readily consented to surgical intervention, and the patient was brought to Louisville the following day.

She withstood the trip well, and after a rest of twenty-four hours was taken to the operating room in St. Anthony's Hospital, and the abdomen was opened by the usual median incision extending from the pubes to above the umbilicus. As the peritoneum was approached, the bluish color which it presented made it apparent that hemorrhage had occurred beneath within the general cavity. When the peritoneum was opened, and the cavity examined, it was quickly determined that the clotted blood immediately beneath was small in quantity and of no consequence excepting to confirm the diagnosis. The tumor-mass proper was composed of numbers of feet of the small intestine and the descending colon, which were densely adherent.

As these intestinal coils were systematically separated, it became apparent that the broad ligament formed the bottom of the sac on the left, the uterus and bladder in the centre, the ascending colon and loops of ileum on the extreme right and on top. In other words, there was a sufficient amount of the small intestine involved to completely cover the entire mass.

Shortly after beginning the separation of adhesions, we were confronted by a rather copious hemorrhage, which increased as the coils of intestine were rapidly separated to enable us to reach the uterine fundus and edge of the broad ligament as quickly as possible, so as to clamp the ovarian artery from which in our judgment came most if not all the hemorrhage. This assumption proved correct, and a well-directed clamp reaching the broad ligament quickly controlled the major portion of the bleeding. The hemorrhage being thus controlled made easier the further efforts at separation of the adhesions, and in a very few seconds we reached a well-formed sac containing about one quart of normal looking amniotic fluid in which was found floating a living fœtus of not less than three and a half months duration. When this was removed and the membranes separated sufficiently to ascertain the correct location of the placenta, it was found that about one inch and a half of the lower edge was attached to the broad ligament, and the remainder of the placenta, which I am sure was not less than four-fifths of the entire mass, was attached to the

mesentery of the ileum at a point about thirty inches from the ileo-cæcal valve.

The question presenting itself for immediate decision was, should we leave this live placenta *in situ* and pack the cavity with gauze to cofferdam the area, according to the teaching in all the text-books of to-day, and allow the mass to slough and come away in the form of a discharge, requiring no less than six weeks for its complete removal, or should we do as has been recently advised by a few abdominal surgeons, attack the mass and attempt to remove at once both membranes and placenta? To attempt the latter method meant assuming the responsibility of controlling the hemorrhage which would almost necessarily be encountered. Immediate removal of the placenta under such circumstances is no small undertaking; in fact, it has been the *bête noire* of the abdominal surgeon for many years.

After a careful survey of the blood supply, which came from the broad ligament and the mesentery of the ileum (especially the latter) I decided to attempt immediate removal, fully appreciating that in doing so the vessels in the mesentery of the ileum might be so injured as to require resection to prevent gangrene. I concluded to attack the placenta from the lower edge, where the clamp had already been applied upon the ovarian artery, hoping that my clamp might control the major portion of the bleeding; if not, there remained ligation of the uterine artery on that side, together with the pampiniform plexus of veins, and finally as a *dernier ressort* ligation of the internal iliac on that side. I was agreeably surprised that only ligation of the veins became necessary to control bleeding from that source.

I then turned my attention to the most delicate part of the operation, *i.e.*, dissection of the placenta from the mesentery of the ileum. To control hemorrhage during this stage of the operation, I had my assistant make pressure on the main trunk of the blood supply reaching that portion of the intestine. After separating the placenta from the underneath side of the mesentery, it was found that no damage had been done any of the larger blood-vessels. A few well directed stitches of fine catgut arrested all hemorrhage excepting slight oozing from the large surface serving as the base for the placenta, which was controlled with hot towels placed firmly against the bleeding surface. After this was accomplished the raw surfaces were coated with liquid

paraffine to prevent if possible reformation of the adhesions. Two cigarette drains, placed against the raw surfaces and brought through the abdominal wound at the lower angle, completed the toilet of the peritoneal cavity. The wound was closed by the tier method of suturing, reinforced by black linen thread reaching beneath the fascia and tied over a liberal roll of gauze.

The time consumed in the operation was one hour and three minutes. I am glad to say the patient made an uninterrupted recovery and went home at the end of three weeks. Inquiry four months later brought the response that she was perfectly well, that she felt no abdominal distress, and had menstruated three times since the operation; that she was doing all of her housework and taking care of her two children.

The details of this case are reported mainly for two reasons: First, the presence of a living foetus in the abdominal cavity, with a placenta and well formed sac attached to the intra-pelvic structures, is of rather uncommon occurrence. Second, immediate removal of the placenta in such cases is a radical change from the teaching and the advice given in text-books. A perfect working knowledge of the anatomy and source of the blood supply to the pelvis and its contents must be possessed before attempting to remove a live placenta. In the absence of such knowledge, or if any part of the necessary operative technic be neglected, serious and many times a fatal hemorrhage may be expected as the most logical result.

The frequency of ectopic gestation is admitted, and instances of abdominal pregnancy progressing to full term are not so rare as was formerly believed. Almost incredible cases are reported in the literature where the foetus remained in the abdominal cavity for long periods of years. It is also known that several successive pregnancies may ensue with normal accouchement during such retention. One instance is recorded in the older literature where an extra-uterine foetus remained within the abdominal cavity forty-six years. In a case reported by Frank H. Yager (*Jour. A. M. A.*, 1912) the patient died after several years' illness, and a foetus covered with calcareous deposits was removed from the abdominal cavity at necropsy. Investigation revealed the fact that thirty-five years had elapsed since the woman experienced symptoms of pregnancy. The foetus may also be retained for a prolonged period in intra-uterine gestation. In one

case reported the time was thirty-two years, the fœtus being partially "ossified" when removed after death of the patient.

The dangers attending abdominal pregnancy were well illustrated by Edward A. Ayers (*Obstetrics*, 1899) in a collection of 148 cases from the experience of one hundred and twenty-seven operators. Of this number 104 mothers recovered, and forty-two died, or a mortality of 40.4 per cent. While abdominal surgery in 1899 can hardly be compared with the excellent work now being done, yet it cannot be denied that the cases herein referred to comprise a most dangerous class which the surgeon regrets to have come under his observation.

The mortality results first from hemorrhage, second from infection, and third from violent efforts to remove the product of conception from the abdominal cavity. Hemorrhage is especially to be feared, for the reason that the site of the placenta is such that ligatures cannot well be applied. Sometimes serious damage to the intra-abdominal contents results from efforts of the surgeon to arrest the hemorrhage by pressure, which is usually obtained by means of firm gauze packing.

Neurology

DIFFERENTIA REGARDING OBSESSIONS AND PHOBIAS WITH REFERENCE TO THEIR PATHOGENESIS AND TREATMENT, SHOWING THE RELATIVELY GREATER IMPORTANCE OF THE MECHANISM THAN OF THE FORM OF PHOBIA * †

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DURING a discussion concerning the management of psychoneurotic individuals, an eminent French psychiatrist showed great astonishment when it was taken for granted by me that not all obsessive phobias were of the hopeless prognosis attributed to them. When it was stated that certain types should be looked upon as rapidly curable, this psychiatrist declared that the differentia of such cases and their mode of management is not known in France and requested that the matter be presented to the Neurological Society.

It has seemed to me, however, that the differentia now to be presented were implicit in modern psychopathological knowledge, and that ever since the epoch-making experiments of Pavlov we were capable of experimental demonstration of the mechanism concerned in the curable type of phobia and besetment.

The fundamental difference between the mechanisms of these two

* In mechanism there is no fundamental difference between a simple obsession and a besetting fear. The primitive genetic factor is not the fear but the circumstantial occasion of which the patient has come to have a dread and which constitutes a morbid occasion feature, as distinct from a normal fear which does not beset a patient.

† Translation from a communication in French made by the author before the Neurological Society of Paris, June, 1918, during his service in France as adviser in Neuropsychiatry for the American Red Cross.

types of phobia is that in the one case we find an emotional predisposition of the patient inherent in the constitution of his organism, which compels him to react unteleologically to circumstances which the average man deals with without serious perturbation. The behavior of the panto-phobiac of this kind is only an attenuated example of the easily excited, uncontrollable phobic reaction of the patient, who is in a state of intoxication such as one so commonly sees in the eruptive fevers, in chronic alcoholism and in the other forms of mental confusion, such as those ensuing upon malnutrition or exhaustion, where the confusion indeed may be very slight, but where the phobic reaction may be most incommoding. The constitutional phobia resembles in its clinical aspect an exhaustion psychosis, so much so that to the condition has been given by one writer the name of "Psychasthenia."

Indeed, clinicians know that the constitutional psychasthenic cannot be cured. All we can do is to alleviate his lot by choosing an environment and by teaching him to avoid what tends to augment his emotivity. But clinicians know that in spite of the best chosen environment these patients inevitably find something about which to show anxiety and concerning which they develop the little manias and fads which are only an attenuated manifestation of phobias and besetments.

It is not these patients, however, that are to be considered in this communication. Very different is the genesis and mechanism of the type of phobia which is so readily removable by present psychotherapeutic methods. This is not a pantophobia, but a monophobia, at least at its commencement. However, such patients tend to multiply the objects and situations concerning which fear or anxiety are felt, so that they eventually wear a clinical aspect which to the inexperienced does not appear different from that of the constitutional panto-phobic. The criterion of difference, however, lies in the genesis of the affective state of the former patients.

The origin of the morbid symptoms can be found by an intelligent anamnesis. They do not proceed from an inherent emotional instability. On the contrary, the patient is, in respects apart from his phobia, usually of a calm temperament. He is even not regarded by his friends as a nervous person at all, and they would be very astonished to hear the history of his inward struggles against besetting fear.

In some cases, however, the patient has no hesitation in speaking of the particular situations which arouse fear. This is when the

patient knows of many instances where fear is aroused in others by the same situation. Especially is this the case regarding fear of high places and irrational fears of the danger of infection by microbes, fear of snakes, the danger of being run over in the street, the danger of going on or in the water. Phobias regarding these situations are so prevalent that few people think of concealing them. Persons possessing these associational fears scarcely suffer morally on account of them, because they easily avoid the situation which provokes them. When they cannot do so, they readily find consolation and sympathy in the common fear of other people.

This fact points to another important element in the symptomatology of the phobic, namely, the wish to conceal his foible from others, his belief that his morbidity is very grave, his dread that his disease will progress to a degree constituting insanity, his fear of discovery. In many cases there is added to these feelings that of shame at his own weakness concerning what he feels to be an absurdity. In such cases the discovery of the mechanism of origin of the particular phobia is an important element in enabling the patient to comprehend the real nature of his condition.

It is only when this is understood he is able to view his reactions rationally, almost impersonally. He learns to see in what way they have occurred, and is thereby enabled to forestall them. This must not be done by a cowardly avoidance of situations which provoke the phobia, but by facing such situations with a clear and open mind and by analyzing his own relationship to the situation each time it arises. In this way, the situation rapidly becomes shorn of its emotional aspect, for the patient has learned to view it scientifically, whereupon the morbid effect which it has formerly aroused ceases.

This method, which I have always found successful, is essentially very different from the former methods by which these patients were treated, such as by emphasizing the lack of gravity of the phobia, by pooh-poohing it, by ridiculing it, or worse still, by attempting to distract the patient in occupations, recreations, or, worst of all, by hypnotism, isolation or rest-cures. These methods, so far from being beneficial are harmful. The rest-cure, for instance, gives the patient more occasion to brood upon his trouble, and even hard work and occupation fail to arrest the morbid process. Indeed, in some instances, intense occupation only gives opportunities for the patient to

multiply the circumstances capable of provoking his phobia, while hypnotism further aggravates suggestibility.

For instance, in one exceedingly hard-working lawyer, his phobia of the number 13 and of the day Friday so fastened itself upon him that there was scarcely any hour of the day which he could not associate with his superstition by methods of addition, subtraction, multiplication and division of numbers, so that the more engagements he made the more he had to struggle against this tendency. In another case, also a lawyer, intense application to study only made more prominent his consciousness of the difference between himself and others.

None of these methods of treatment aim at the cause of the condition, as all medical art should. The *essential cause of phobias* of this type is a *conditioning of the effective reaction* towards a given situation *because of a mistaken notion regarding it*. The mechanism is most simply and clearly illustrated by the reactions of the animal in whom a given signal has always been associated with a given experience. In the course of time, the signal alone is sufficient to induce the effective response capable of being produced by the experience itself. Pavlov found that dogs whipped when a bell rang learned to cower with fear of the bell itself, in anticipation of the painful experience. He observed that dogs fed at a certain signal would secrete gastric juice as soon as the signal was given, thereby anticipating the flow which usually occurs only when food is seen or smelt. Furthermore, although these responses are entirely involuntary, depending as they do upon the vegetative nervous system, so manageable are they that they can be reconditioned, i. e., the same signal which at one time provoked fear can be later utilized to provoke pleasure if the dog is reeducated by accustoming him to associate with this signal a pleasurable experience, whereby there is a gradual disappearance of the painful or fearful association.

It is by the use of this principle of reassociation and substitution, performed, however, with the deliberate understanding of the patient, that we effect the disappearance of phobic reactions to environment.

Long ago Janet outlined the principle of substitution in the removal of hysterical obsessive ideas. The present writer dwelt at length upon the principle in his study of intellectual precocity (Pedagogical Seminary, 1909), where he showed the necessity of substitution for motives which promoted disinclination other motives which

gave a taste for the studies towards which it was desired to direct the pupil. Janet's classical case is that, where during hypnosis, he substituted for the head of a handsome young man which obsessed an amorous young girl the head of a pig, whereby a feeling of disgust replaced that of attraction ("Névroses and Idées fixes," 1898).

The same principle has been set forth under the name of "The Setting of Ideas," by Morton Prince (Presidential address, American Psychopathological Association, 1914). It has always been recognized by the great poetic dramatists. Thus, Shakespeare makes Hamlet say, "Nothing is but thinking makes it so."

In these days the principle has been objectivated, so to speak, experimentally even upon animals, by the immortal work of Pavlov, who, by substitution, added to and subtracted from the meaning of certain stimuli to which he subjected his experimental animals, whereby he completely changed their reaction to the stimulus, as by inducing a flow of gastric juice upon the ringing of a bell, and by inhibition the flow through the presentation to the animal of a whip. But these stimuli did not act until the animal understood the significance of the objects presented. Of course, these experiments are merely the restatement in precise physiological terms of phenomena with which every animal trainer has been familiar for generations. Mark, however, that they express in simple, seemingly non-psychological reactions, the very basis of all psychotherapy.

The essential of all this is the change of the subject's notion as to the meaning of the situation which provokes the emotion. THE EMOTION CANNOT BE CHANGED UNTIL THE SUBJECT ENVISAGES THE SITUATION DIFFERENTLY. In other words, the phenomena depends upon perception, observation, cognition, intellection. In his studies of the mechanism of traumatic neurosis, the author has shown this very clearly and at length. (See "Affective and Intellectual Processes in the Psychoses Known as Traumatic Neurasthenia," *Journal of Abnormal Psychology*, 1910; do *American Journal of Medical Sciences*, 1913; do *Journal of Criminal Law*, 1916; "Traumatic Neurosis and Babinski's Conception of Hysteria," at the International Congress of Industrial Accidents, at Rome, 1909, and *Medical Record*, 1909.) The mechanism of induction or conditioning, by means of which the affectivity is influenced through the perceptions and understanding, is an entirely similar mechanism, which has been illustrated by the

author's "Psychogenetic Disorders in Childhood" (*Journal of Abnormal Psychology*), and in "Psychotherapeutic Symposium" (Gorham Press, Boston, 1909); also in "Juvenile Psychasthenia" (*American Journal of Medical Sciences*, 1912), and "Mechanism of Hysterical Phobias in Childhood" (*Medical Record*, 1910).

In all of these studies it is shown that the unusually rapid removal of the disturbances of the patient, which in those days appeared astonishing to other physicians, was merely due to the very clear keeping in mind of the principle that the emotional disturbance was not due to vaguely envisaged hereditary constitution, not to a specially "nervous" disposition, but was traceable to a faulty attitude of mind or way of looking at the situation, of which the phobia, obsession, tic or what-not was a reaction, and that *reconditioning of this attitude was a purely intellectual process*, and invariably brought with it the alteration of the affectivity.

It may be objected that the dogs of Pavlov were subjected to an artificial experience over a relatively short time, and that the laws gained by their study cannot be applied to human beings who are subjected for a long period to the ordinary circumstances of life. True, it is a clinical commonplace that the psychoneurosis which has lasted a long time is harder to deal with than one of recent origin; but that length of time is no bar to successful treatment I have proved, and the accompanying instances clearly show it. These instances are:

AGAROPHOBIA COMBINED WITH CLAUSTROPHOBIA REMOVED IN ONE WEEK

CASE I.—A woman of thirty-three years, whose sister had been sent to me on account of an intense migraine, consulted me because of her inability unless accompanied to cross a wide street or to remain in a church or theatre, without an intense emotional disturbance, showing itself as palpitation, polypnoea, facial pallor, chilliness, moisture and cyanosis of the extremities, rigidity and pain in the neck and back, nausea, the sensation of great weakness and dizziness, which had lasted for eight years.

Examination showed no physical disease other than myopia, rather prominent eyes, slight fine tremor, excessive sweating and an enlarged thyroid, instability of the pulse rate and of the blood-pressure. The latter at the first examination was 160 systolic, 100 diastolic, while

the next day it had reached only 140 and quickly fell to 127 systolic and 85 diastolic, which I regarded as her normal pressure. The pulse rate, which at first had been 112 per minute, later was found to be 97, and was reported to me to be habitually less than 80.

The patient declared that while outside she was never able to relax, and had lately been becoming apprehensive even in the house, and had also felt very weak in the evenings, when her heart would often flutter apprehensively.

It is not because she dreads an accident that she cannot cross the street alone, but because she fears losing consciousness on account of the heavy sensation of oppression which she experiences.

She was asked to recall the first occasion upon which she had experienced these sensations. After some effort she was able to recall that in church eight years before, on a hot summer day, she had begun to feel an overwhelming sense of illness during the sermon. The compulsion to leave the church was intense, but she was ashamed to do so as she sat near the front, well back in the pew and did not wish to excite attention by creating a disturbance. She had not been ill at the time; indeed, was an exceptionally strong girl; had had no worries, and the subject of the sermon caused no painful impression upon her. No attack reoccurred for some weeks. At the end of that time another attack did occur, and gradually she began to experience these incommoding symptoms either at church or in the theatre, and she was only able to avoid them by sitting near the door, so that she could get out immediately she began to feel oppressed. About four years later, she began to fear crossing a wide space, and she felt the need of someone to support her.

She had always had a fear of high places, but had thought nothing of it, as everyone else in the family felt the same way. She had never feared the dark, and she had no social timidities, and had enjoyed her school and college life. She had always been anxious about the health of her mother, who was an invalid, but did not reproach herself for this, as she had always attended to her. As a girl, however, she had been timid about appearing conspicuous, as in recitations in class.

A series of association tests were made, but revealed no morbid effects, except when direct leading words were used, such as the "pavement."

Further interrogation brought out the fact that it had been a very

hot day when the first attack had occurred, in a small, ill-ventilated country church, and that she had really experienced a physical oppression which was antecedent to the moral distress at the possibilities which she imagined.

It was concluded, therefore, that the hyperthyroidism evidenced by the size of the gland, the hyperhydrosis, the prominence of the eyeballs, slight tremor, tachycardia and mobility of the pulse and blood-pressure was not the most important feature of the case, and might, indeed, be a condition secondary to the chronic emotional strain to which she was subjected, and that it might disappear if this were alleviated. Further, it was believed that hyperthyroidism could not be responsible for the emotions of the patient, for the psychic reaction was so specifically contingent upon definite circumstances, whereas the hypermotivity of hyperthyroidism is occasioned by numerous circumstances, and does not tend to fix itself upon only a particular event.

It was, therefore, concluded that both the agoraphobia and the claustrophobia which this patient experienced were hysterical notions arising from the powerful suggestion of the recollection of a particular experience which was efficacious, now only by intermediary of the timorous imagination of the patient.

Accordingly, reëducation was forthwith begun. The patient was at first unwilling to undertake it after my explanation, giving the excuse that she had never been willing to introspect; but she accepted the necessity of doing so when it was explained that a person was under an obligation to know himself, and that it is as futile to oppose this need as it would be to object to understanding technic if one were learning to play the piano.

It was explained to her that her dread of what might happen in a close or open place was merely due to her own ignorance of the mechanism of the consequences of a wrong way of looking at things and the emotions brought about thereby, and that only when she obtained a true insight into her own psychological machinery would she be able to control it. The power of induced ideas and the feelings produced thereby were explained to her and illustrated by the story of a play called "The Harvest Moon," in which a hard-headed lawyer was made sick by the means of a few words skilfully implanted.

After a while she accepted my explanation, and added: "It must

have been fear, because on leaving the church, one of the maids said, 'What frightened you?' "

She stated that she was relieved after the relation of her feelings to me because her sister had maintained that she had been wrong in showing too much sympathy for the patient's affliction. She added, too, that she was naturally "such a self-contained person."

In the course of a few days she was asked to write her account of the way in which she viewed her own psychology, and this I append:

"After several years of intense suffering from supposedly physical causes, it is, to say the least, surprising to be told that I am the victim of fear, and that the fear recognized and removed, the physical symptoms will disappear. Of course, I know abstractly that fear is psychological, and that it does produce physical reactions, just as other emotions do, but I find it hard to convince myself that the fear of fainting on the street or in an audience, is, in my case, the source of all the unpleasant and peculiar sensations I have in such a marked degree. It still seems to me that there must be something other than this vague intangible 'unknown quantity' that makes me tremble at the mere thought of walking a block or two. And yet that line of argument brings me back to the psychological phase, for I have spontaneously written that I tremble at the *thought*. I have almost resented the idea of anything psychic, for it has seemed to me like an admission of weakness or lack of will power, but I believe I am gradually coming to see that mind has something to do with matter, even in my own case, and that distant as it seems now, the physical symptoms so uppermost and persistent may yield when I fully grasp the fact that there is nothing to fear.

"A few days ago I heard what seems to me to be a very striking illustration of association of ideas. A man who had been in the trenches was riding on a trolley car here in Washington, when the current was short-circuited and the lights in the car suddenly went out, with the noise and buzzing sound that accompanies it. Immediately the soldier fell flat on the floor of the car, but before anyone could reach him, he had arisen and seemed perfectly well. He then explained that ever since hearing the explosion of bombs and bursting of shrapnel in the trenches, any unusual sound or sudden noise produced the same shock, and wherever he happened to be, he instinctively threw himself flat on his face, for protection. This is, of course, an extreme

case, but it is true that to a greater or less degree impressions are being constantly induced upon the human mind and no one can escape the influence of them. One of the most familiar instances is that of actual illness produced in a perfectly well person by the power of suggestion which is so clearly brought out in the case of the lawyer in the 'Harvest Moon.' I have seen a happy, smiling baby draw down the corners of his mouth and finally cry real tears because someone had used a sympathetic pitying tone of voice in speaking to him.

"The casual greeting 'How are you?' sometimes sets up, in a sensitive person, a train of thought that brings with it a whole list of physical ills not thought of before. Much has been said lately about the *moral* effect of the presence of American troops in the European trenches aside from the real reinforcement of man-power. Bishop Brent insists that the mere appearance of the American Flag on the battlefields of France, no matter how small a force of men accompanied it, would be sufficient to hearten the whole French Army and send it on to greater victory. Instances of *the power of an idea* might be multiplied, but it is evident that the human mind is easily swayed by impressions. It seems to me important, then, to see just what sort of big ideas are dominating our lives and giving color to our work.

"About eight years ago I had an experience which marks the beginning of a definite period of my life during which I have suffered in the most peculiar and distressing way. As I try to recall it now, I have a very clear mental picture of the church where I was at service, and also a very vivid recollection of the sudden overwhelming sense of illness and oppression and a great desire to get out into some less restricted place. However, with a great effort I did stay until the end of the service. I thought little more of it and attributed my discomfort to some slight physical disorder. But ever since that morning I can truthfully say that I have never been in any public gathering when I felt at ease and thoroughly relaxed. Always I am restless and uncomfortable and fidget about, waiting anxiously for the moment when I can be free from the strain. Even though intensely interested in sermon, lecture or play, and regretting to miss a word of it, I am often so really ill that I have to leave quickly for fear of fainting or making a scene of some sort. I remember that several years ago a certain magazine published views of the interiors of the great opera houses of the world. Absurd as it may seem, those pictures were

frightful to me. I could not look at them without having the same sensations of space and height that I knew so positively I should feel were I actually sitting there in the flesh. This is to me pretty convincing proof that the idea of fear is the real cause of all the symptoms which have become more and more exaggerated until at times I am really ill.

"The same dread or fear of space it must be that makes me feel uncomfortable and unnatural when on the street, for I seldom walk half a block without swaying and dizziness and the certain conviction that at the next step I *must* fall.

"This fear, in my case, was evidently set up that morning in the church, and ever since I have supposed I needed medical treatment for some serious trouble, but now with the assurance that there is absolutely nothing wrong in my physical make-up, my problem seems to be to rid my mind of the fear that has unconsciously, but, as I see it now, completely controlled my thought and made the days when necessity urged me out of doors or my desire led me to some public function, occasions to be dreaded beforehand and looked back upon with horror.

"It is difficult for me to understand that these signs of illness are not illness at all, but caused by an induced impression—but I have high hopes that this view of the case is the final and correct one."

In the meantime she had complained of pain in the sacrum, and wished to make sure that there was nothing physical to cause it. She insisted that I examine her. I found no tenderness even on rotation of the hip, and only a slight scoliosis. There was no dilatation of the colon, and the appendix vermiformis had been removed. The surmise that the pain was due to the postural dragging induced by the restricted manner in which she walked on account of her dread was confirmed by the fact that it disappeared when she adopted a freer manner of locomotion.

The next step in the treatment was to accompany her to a large square in the neighborhood, across which I made her go alone. Although her hands became cold and her face pale, the pulse frequency increased and her throat became dry, she declared that she had performed the feat better than she had done so. The following day she had to do so on several occasions alone.

When she came to see me again she declared: "I can't get over it. I feel so different, but I dread the return of the trouble. The day

after you led me across the square was the best I have had for years. I went to church and enjoyed the service and experienced no palpitation. For a moment a sudden fear appeared, but I stopped and reasoned concerning it and concluded that nothing could happen." I concluded with the final adjuration that all now depended upon herself, and she realized that she was well. She remained so a month later. I have not heard from her since the war; but a relapse need not be feared, as in ten years I have never encountered one in a patient treated in this way.

Commentary.—Thus, violent, persistent, long-continued agoraphobia and claustrophobia were traced to a single incident upon which they were dependent. They were removed in less than a week by efforts directed towards giving the patient an understanding of their mechanism; indeed, compelling her to grasp it, and then compelling her to take an exercise which afforded a practical demonstration.

LONGSTANDING FEAR OF DEATH REMOVED IN A WEEK

CASE II.—The rapidity with which the genesis was discovered in this case is not singular; in fact, I have found it the rule. Thus, during the past spring, a man, aged thirty years, was referred to me by Dr. Sterling Ruffin because of a nervous breakdown.

Superficially and in his own estimation he appeared to be simply agitated, terrorized and quite incapable of conducting the business which had brought him to Washington.

His face was congested, eyes bloodshot; he trembled violently. Indeed, the friend whom I first saw, doubted his willingness to see me. In spite of this, a single interview sufficed to discover that this abject picture was not caused by physical disease, but was the result of phobias. The origin of these was ascertained in part at the same interview, and during the following few days the genesis was penetrated adequately to permit the man to return to his Western home, no longer incommoded by the morbid fears which had incapacitated him.

This man's fear had originated during the excitement and horror caused by the bursting of a large dam in the city where he lived. His efforts to reach his home and rescue his mother were impeded by the crowd, whereby an anxious terror was induced. This persisted as a fear of water and of storms. Eventually the wind became the most conspicuous feature which aroused fear. Fire would also do this.

Recently the phobia was becoming associated with circumstances more and more numerous, and he had become less and less fit for his business, which was of an exacting nature. He had been sent to Florida for a change, and while there got better. He quickly relapsed, however, upon returning to work.

The real meaning of these phobias was fear of death—and it was this that had to be dealt with before his peace of mind could be restored.

It was very necessary in this case that each step he took should be expressed by him in writing before my review. It was in one of these exercises that he made the significant declaration: "Perhaps I don't amount to much, anyway." He made the further statement: "I have concluded I will be just what I make myself."

This patient was the antithesis of some of those which follow in that so far from having a sense of inferiority, he felt himself to be a highly superior individual. He was an only child, spoilt, and things had always gone well with him, even in business, where he was protected and almost petted by the heads of the firm. It was not until the sense of humility was aroused that he was able to deal with the groundwork of his fears.

MORBID ANXIOUSNESS ABOUT SUFFICING FOR TASKS

CASE III.—A feeling of inadequacy or inferiority is a dominant state in some individuals. And these need not be constitutional inferiors in deed.

The processes through which the child goes during education for civilized life raise a constant presumption of his inferiority. This is especially the case in the more stable societies, where conventionalism is highly developed. The feeling of inadequacy, however, may not beset the individual and life may pass relatively comfortably in one who sets himself willingly for an inferior rôle.

One of the most curious turns to a besetment of this kind which I have encountered, was that of the woman obsessed constantly by the idea that she was necessarily inferior to the men around her because she was a woman.

This not only made her very unhappy, but interfered with her sufficiently and disturbed her social relations with men also in the bureau where she was employed. The idea, however, remained an

obsession and never became fixed into a delusion; she recognized it as an erroneous way of looking at herself, and yet she could not shake it off, because the intellectual inferiority of women had been inculcated from her earliest years, and more particularly when she grew up by a near relative whom she highly respected.

My management of this obsession consisted of a process of gradual enlightenment regarding the anatomy and physiology of the nervous system, along with instruction regarding the fundamental differences between man and woman. By this means the patient was able to orientate herself truly in life and ceased to be obsessed by her highly inconvenient idea.

A fruitful source of morbid fear of inadequacy is, of course, frequent failure. Many a person is inefficient only because they were early in life set tasks without proper instruction in method. It would take too long here to discuss in detail the psychology of this kind of obsession, especially as I have very fully discussed its mechanism in a previous publication (*The Genesis of Intellectual Precocity, Pedagogical Seminary, 1909*).

There is a temptation to diagnose cases of this kind as constitutional inferiority. This is a pitfall of those who perform their diagnoses mechanically by the enumeration of symptoms. This error will be avoided by those who make diagnosis of means of the valuation of processes, not only psychological, but physical as well.

The avoidance of this pitfall is well illustrated by the very remarkable case which follows:

SOI-DISANT PANTOPHOBIA, WITH BAD HEREDITY, IN REALITY PRODUCED
BY FAULTY TREATMENT IN INFANCY, CURED BY RE-EDUCATIVE
PSYCHOTHERAPY

CASE IV.—A professional man, twenty-eight years old, became so beset by nameless fears which so worked upon him that he gradually withdrew himself from society and friends, abandoning work and food, finally commencing two attempts at suicide. He was quite unable to define his besetting phobias, and their manifestation was the cause of a great deal of secret shame. His struggles dated from his college days, and he had made many attempts to obtain a mastery of himself by means of games, and had even spent six months later on in a camp of rough lumber men as a laborer, in the hope that the ex-

perience would prove beneficial. His experiment failed, whereupon he plunged into study and office work without ever obtaining the relief he sought, until after several consultations and six years of ineffectual struggling he was brought to me.

Physically, the only important features he presented were a great loss of weight and a high degree of erythism. Psychological exploration was begun by my asking him to search his memory for fear-bearing experiences dating from his early life, the elucidation of which might lead to a removal of the cause of his trouble; but he could think of none. Finally, the patient declared that he seemed always to have been afraid, and that he feared he must be a physical degenerate. He also thought that heredity might have something to do with it, as he had a brother who was a wanderer over the face of the earth.

It was not until the remembrance of a near relative was evoked that the key to the situation was found. It appears that this individual had been a believer in the hardening process for the development of the physical and moral welfare of youth, and the manifestation of this theory had led to such procedures as throwing the boys into water when unable to swim, fishing them out only when almost breathless. In winter, they were thrown while asleep into a bank of snow, after which they were snowballed home to the door. Another procedure was to chase the children with a stockwhip from the door to a tree in the distance. Far from hardening these children, these manoeuvres only succeeded in producing chronic fear. The recollection of these performances dated back to the age of four, but the patient had completely put them out of mind and believed that his fears had originated in the high school.

It was then explained to him that his cowardice was merely a psychic habit and not an instinctive reaction, and that intelligent effort would remove it. It was necessary first of all to ensure several days of regular eating and sleeping, and to this end I hypnotized him to sleep, after which he carried out a dinner programme we had arranged. This was done three times in all, but not on consecutive nights.

In the meantime reëducation was begun. This consisted of a reconstruction of the fear situation of his infancy, and of an insistence upon the possibility of a readjustment of his reactions towards himself and the world. After four days' treatment I left him, but a friend drawing my attention to a relapse, relations were again resumed.

After four days more, the tide turned, and he obtained control of his fear. Later, he gave me a lively account of how he overcame a facetious waiter he had encountered in a restaurant a week previously. His unhappy appearance had excited the risibility of the waiters, and upon the second occasion of his visit there, one of these came forward grinning. "I looked him in the eye," said my patient, "and the grin changed to a smirk. I kept him standing waiting while I read the menu through, and I said, 'Bring me this and this and that, and, waitah, hurry, and don't you dare not to do so always.'" The patient has since led an active professional life; later married; remains well seven years later.*

PHOBIA OF THE DARK REMOVED BY RE-EDUCATIVE PERSUASION AFTER
SHORT ISOLATION

CASE V.—This was the case of a girl sixteen years, who would frequently wake in the night very much afraid unless she was soothed by someone sleeping with her. She had been much spoiled, owing to a supposed weak heart, and had always been considered delicate. Her father and aunt had been timorous as children, the latter for nine years had not dared be alone for a moment. Inquiry showed that a servant had told terrifying stories to her sister as a child; the horrors of this had run through a family of three children, but had passed away from all but this patient. Her fears were either of fires or burglars, and they only occurred when in bed or asleep. Upon waking in a fright, she would be reassured by the touch of a companion. She could not remember the first occasion of fear. The creaking of the floor would make her think that there was someone in the house, although she knew that positively this could not be the case. Analysis showed that in the case of her fear of a burglar, the fear was rather of the unknown than of anything specific happening to her. She had been accustomed to go to sleep with her nurse in the next room. If she woke up in the middle of the night terrified, as she usually did, she would go into her mother's bed in the next room. It was only during the last few months that her mother had been sleeping with her in the same room during the entire night. She was always more afraid in their city home than in the country, as she thought that the danger from burglars was greater. Even in the country, however, al-

* See "Genuine vs. Spurious Psychotherapy," *Illinois Med. Jour.* (1914).

though she would get to sleep more quickly, she would always have someone with her. In addition to all this, she was greatly ashamed of what she called her "silly babyishness."

Three dreams were obtained. The first and second were of a burglar entering the window. The analysis showed only that the intruder aimed to shoot her sister who was standing behind her; a dream of fears of elevators led to no pertinent associations. The dream analysis being so unfruitful, it was thought better to proceed at once to the re-conditioning of the psychological reactions. This was attempted in the first place by giving her something to read about the psychology of fear, and explaining to her what she could not understand alone. In the second place, she was given exercises in mental concentration, and with increasing proficiency in these was urged to apply them to the study of her own feelings of nocturnal apprehensions, the principle she was made to grasp being that the fear and shame of her fears prevented her from facing and examining them, which was the essential preliminary to the understanding which would make them disappear. In ten days she was beginning to obtain mastery of the fears, but was still unable to sleep alone. A month later her mother wrote me that she was entirely well, and when she awakened in the night, would quietly turn over and go to sleep again without troubling anyone, and was in better health physically and mentally than at any time in her life. The patient has remained well for six years.

OBSESSION OF INFERIORITY LEADING TO FOUR ATTEMPTS AT SUICIDE,
CURED BY RE-EDUCATIVE PERSUASION WITHOUT ISOLATION

CASE VI.—This was a farmer's son, aged twenty-two years, who attempted to throw himself into a creek. Rescue and reproaches on the part of his brother failed to deter him, for a few weeks later he swallowed laudanum. Removal to a sanatorium prevented a fatal result, but he made two further attempts at suicide, until he finally was brought to me. Examination showed no physical disorder, although there existed a serious psychological situation which had been quite unsuspected.

The management of his mother's farm had descended upon this boy consequent upon the death of his father, but he had been greatly mortified by the interference of a younger brother, with his plans,

His mother also acquiesced with the meddling of some neighbors, and the situation became intolerable to him. He also complained of stomach troubles, although he had no pain. In reply to a further question he stated that the reason he took so much laudanum was that he thought he would be better dead. He said he could not stand being worried by his family, who seemed to find fault with everything he did. The reason he took everything so hard was that they said he had poor judgment. With regard to his relations towards the opposite sex, he had decided that on the whole girls were not worth bothering about, although he had liked their society. He had never cared for any particular girl, although he had desired them. He was naturally very shy, a fact which provoked feminine laughter, and the boys laughed at him when he told them that their indecent talk was wrong. He had dreamed of erotic situations, however, and had provoked diurnal emissions until he was eighteen, after which he had ceased to do so, as other boys often teased him about it and had told him that he would be impotent, as he had ruined himself, whereat he was much ashamed.

The interpretation of this case was that the failure of this boy to stand up for himself was his own shame at a mental inferiority which he feared had been caused by his own onanism. This shame added to the teasing to which he was subjected from other boys, caused him to withdraw from social life. He confessed that if he could be cured of this hopeless mental inferiority he would be willing to live and would like to work.

The treatment consisted in assurances, with examples given to show that he was mistaken about the effects of onanism; and he was asked to think over until the next day the explanations I had given him concerning the genesis of his shame and timidity, meanwhile promising not to attempt suicide until he had seen me again.

The next day the discussion was resumed, until in less than a week the boy could be trusted alone, not only in the grounds of the hospital where he had been taken, but in the town. He went home in ten days completely cured. The pains referred to the stomach, which, of course, disappeared, were merely an attempt at fixation of his discontent upon a bodily symptom. He has been at work and in good spirits ever since. The treatment was conducted in a general hospital and the maximum of freedom was allowed the patient from

the first, the greatest tact being urged upon those who nursed him. (See "Prevention of Suicide," *Trans. Am. Medico-Psychological Association* [1914]; also *Jour. Social Med.* [1917].)

THE PREVENTION OF PHOBIAS IN INFANCY

CASE VII.—This illustrates the arrestation of what would have become a night terror if injudiciously handled in the case of a boy aged three years and nine months. For several weeks he had been visiting the zoological garden every afternoon in company with a French maid of exceptionally forceful character and apparently free from the superstitiousness of the average nurse. One evening he began to cry soon after he was left for the night. When questioned about the unusual occurrence, he said that there were lions in the house and that he did not want to stay alone, as he was afraid they would eat him. It appeared that the lions had roared more loudly than usual that afternoon, at which he had been much impressed, although untterrified. The boy was soon convinced that the lions must stay in their cages, and that, therefore, there could be none in the house. The sense of security gained by a little affectionate demonstration was first obtained, and then conversation was carried on by talking about something else. In this way the state of terror was dismissed, and instead we joked about "the funny roaring of the lions" before we ceased talking; so that finally the little fellow lay down with the solemn purpose of going to sleep and thinking of the cars and motors passing outside his open window. Thus through simple substitution for what might have been a serious fear psychosis was prevented.

Fear of the water, so common in children and women, can be not only prevented upon these principles, but can be as readily removed as any others of the numerous phobias and besetments.

These are only a little more difficult to get rid of than were the anxieties and dreads of soldiers at war. Our psychiatrists in the army proved to the last the practicability of dealing successfully with these. It is time that not only more surgeons, internists and other specialists should realize the curability of these patients, but that the public also should be enlightened as to our power, and that they should be convinced of the damage of consulting charlatans when their psychic health is concerned.

MEDICAL SUGGESTION AS CAUSE OF DISEASE

CASE VIII.—Some varieties of phobia arise from suggestions of medical origin; and in many instances morbid fears are maintained by the attitude of medical men of whom advice is sought.

This is particularly true when the heart, stomach and the generative organs are concerned. The danger is greatest when examinations or treatment of a case is accompanied by much chemical analysis, many clinical explorations by imposing apparatus or much surgical manipulation.

The neurologist who sees these cases after mismanagements of these kinds has to studiously avoid any procedures which may seem to the patient to maintain attention upon the organ concerning the function of which the phobias exist.

Thus, in a case seen this spring where fear of loss of sight had accompanied a ptosis of psychogenetic mechanism, it was necessary to sedulously avoid even examining the patient after the first diagnosis had been made. Even visits were studiously neglected as one of the means of persuasion that there was not any condition demanding surgical or medical procedure. The taking of this precaution enabled a cure to be obtained in a few weeks in this case, which might be labeled either palpebral tic or hysterical ptosis. The condition had been greatly aggravated by the frequent repetition of examinations—ophthalmological, psychiatric, medical, neurological, otological, serological, endocrinological, and so on.

None of these had sufficed to ascertain the genesis of the tic, which was readily done by an intelligent anamnesis in conjunction with a study of the actual character of the ptosis. The falling of the lids occurred irrespective of fatigue, in the absence of conjunctival irritation, without the least sign of facial paralysis nor other organic nervous disease. Although the closing of the lids was usually imperative, yet they could sometimes be opened spontaneously by different devices.

This is characteristic of every tic. However, the more the patient wished to open the eyes, the more tightly and longer they closed.

At first it was only by methods of distraction that they could be opened by me. These, of course, were used merely for diagnostic purposes; for therapeutically they are inefficacious. The patient had

already discovered this after the eventual failure of such methods as clacking the tongue, coughing, whistling, etc.

The genesis was interpreted as follows:

The conjunctiva had become irritated by considerable driving along dusty roads in an automobile in the glare of the summer sun, in conjunction with the short sleep and the conviviality entailed by late hours.

The protection of the eyeballs by lowering the lid was the consequence. In the manner of the tics, this psychological response eventually became a psychological habit. This habit the patient might have shaken off as he had done previously with other tics; but in consequence of repeated medical opinions expressed during six weeks before him by distinguished men in a famous hospital nearby, there was added to the habit the phobia of the inability to open the eyes and the fear of loss of sight, and with it, earning capacity.

PANTOPHOBIA

The conditions we are discussing are very different to that seen in the pure fear state—pantophobia. In persons thus suffering there is a profound disturbance of the physical organism. This is usually toxic, or, at least, chemical in kind. It is merely a state of painful affectivity, due, either to an erythism of the central apparatus, or caused by an excitation of the peripheral receptors.

French psychiatrists have long supposed that these are in the main situated in and around the great viscera. They have termed the disorders of this kind cenesthopathias, by which they mean, a disease of the common body, internal sensibility as distinct from the external sensibility comprised in what have been known as the various senses.

Protopathic sensibility is the name given to a similar concept by other clinicians, such as Henry Head. Still more recently we have learned to connect the excitation of these receptors (which are subserved, of course, by the autonomic nervous system, including the sympathetic) with modifications of the internal secretions. In turn, we trace endocrin disturbances to excitations and inhibitions of this vegetative nervous apparatus. Nowadays, the inductions of what has been named visceral neurology are influencing a good deal

of clinical investigation powerfully tending in the direction of chemistry through the theory of the hormone.

Cenesthopathic disturbances, not only produce local discomfort, but strongly influence the personality and consciousness of the sufferer. The sensations are often so peculiar that although within they cannot be reconciled with the usual feelings and the accustomed concept of the patient's selfhood. The realization of this incompatibility sometimes engenders fear on account of the inference that something is going seriously wrong. This is especially so when the degree of strangeness is such as to bring the idea to the victim that he is losing his mind. This is not an uncommon mechanism in that very common phobia.

I have not related a specific case where fear of insanity was the dominant phobia. Not only is it present in so many, but it is a fear that should rarely be treated directly. In this respect, it resembles a so-called psychalgia or hysterical pain. The best way of getting rid of this is to ignore it and deal with the mechanism of which it is an indication. Fear of insanity, too, is very easily disposed of when the more fundamental factors of which it is an expression, are properly dealt with.

Cenesthopathic sensations of physical origin are themselves an entirely different problem. Medical means must be sought. The surgical measures which have sometimes tempted the enterprising, as when a patient believes there is a parasite gnawing at the vitals, are to be avoided; for in the true cases the sensation persists in spite of the operation. In the few instances where the operation has removed a visceral paræsthesia, it is a hysteria that is being dealt with and not a cenesthopathia at all. Even here psychotherapy would have been infinitely preferable.

Psychic means, of course, are inefficacious against organic sensations of physical origin. I mention these cases only that they may be in mind when the false pathias, psychogenetic in kind, have to be considered.

Pædiatrics

EDITED BY JOHN FOOTE, M.D.
Washington, D. C.

CLINIC OF DR. JOHN LOVETT MORSE

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TUBERCULOUS MENINGITIS

October 17, 1919.

THIS little boy, Samuel S., eight years old, was admitted to the hospital, October 15, 1919. His father died of tuberculosis. Unfortunately the house-officer neglected to find out when he died and whether or not the boy was exposed to infection from him. His mother, one brother and one sister are alive and well. There have been no deaths or miscarriages.

He was born at full term, after a normal labor, and was normal at birth. He was nursed for a year and then given the regular family diet. He has always been well, except for mumps last August.

He was taken sick suddenly October 6th with frontal headache and pains in the neck. He felt so badly that he went to bed, where he has been ever since. He vomited three times October 8th, each time, however, after a meal. His bowels have been constipated, but have been moved with enemata every two or three days. He has craved for various foods, but has eaten very little. He has been very drowsy most of the time and has slept a good deal. At times, however, he has awakened and complained of severe headache. He has been very irritable, and disturbed by the slightest noise. His mother thinks that he was not feverish at first, but that he has been somewhat feverish during the last few nights. Day before yesterday morning he had a slight nose-bleed and, when he entered the hospital in the afternoon, there were small clots of blood in each nostril and streaks of fresh blood in the pharynx.

The onset with headache, followed in two days by vomiting after meals, suggests a simple attack of indigestion. The persistence of

the symptoms of headache and drowsiness, without further vomiting, show, however, that his trouble is something more serious.

The combination of the onset with headache and pains in the neck, followed by drowsiness and irritability, and the persistence of the headache, points very strongly to some cerebral disease. The vomiting is consistent with such a diagnosis, although cerebral vomiting usually does not occur immediately after eating. If his disease is cerebral, it is, of course, acute. Chronic conditions, such as tumor and hydrocephalus, can be excluded by the sudden appearance and rapid development of the symptoms. The forms of acute cerebral disease which may be reasonably considered are meningitis, the encephalitic type of infantile paralysis and acute encephalitis, not connected with infantile paralysis. The onset is consistent with all of these diseases. The persistence of the symptoms for nine days, however, without much improvement, together with the fact that the boy is here to be shown to you, point very strongly against either form of encephalitis, because, if either of these conditions was the trouble, he ought to be dead, much worse, or much better by this time. Furthermore, there is no epidemic of infantile paralysis in the community. These points, however, do not absolutely exclude these conditions. Meningitis is, therefore, the most probable diagnosis. Syphilitic meningitis is not at all probable, because syphilitic meningitis does not ordinarily develop acutely in a child of this age without there having been a story of other syphilitic manifestations previously. If the boy was a baby, syphilitic meningitis would have to be considered more seriously, because acute syphilitic meningitis is much more common at this age. Meningitis due to the influenza bacillus, the pneumococcus, or the pus organisms can be excluded, because influenzal and pneumococcal meningitis always develop secondarily to influenza or pneumonia, which the boy has not had, while the meningitides due to the pus organisms always follow some local infection, which he has not had. If he has meningitis, therefore, it must be either tubercular or meningococcal. The acute onset is in favor of the meningococcal type, but the persistence of the symptoms, without marked increase or improvement, is in favor of the tubercular and against the meningococcal form. There is nothing about the symptoms themselves which is of any assistance in differentiating between these two forms.

The nose-bleed, which he had day before yesterday, opens up a new point of view, however, because nose-bleed is not a common symptom in either of the types of meningitis which we have been considering. It suggests at once the possibility that the trouble is typhoid fever rather than meningitis. The headache, drowsiness and irritability, as well as the vomiting and constipation, are perfectly consistent with this diagnosis. Pain in the neck is not usual in typhoid fever, but may be simply referred pain, as children are often unable to locate pain accurately. The onset may seem rather acute for typhoid fever, but the symptoms of typhoid often appear quite suddenly in childhood. It is possible that their sudden appearance is only apparent and that the child has not mentioned them or given in to them until they were so severe that he could not resist them longer. Nose-bleed is, however, a relatively uncommon symptom in typhoid fever in childhood. It, therefore, does not point so strongly to typhoid fever in this instance as it would if he were an adult. While, therefore, typhoid fever is a possibility, it does not seem so probable as one of the forms of meningitis.

His temperature, since he has been in the hospital, has ranged between 101° and 102.4° F. His pulse has ranged between 72 and 82, and his respiration, between 20 and 28. The temperature does not help us at all in the differential diagnosis between typhoid fever and meningitis, or between the two types of meningitis, since it is consistent with any of them. The pulse is relatively slow in comparison with the temperature. This again is consistent with either meningitis or typhoid fever. It points rather more strongly, however, toward typhoid fever than meningitis, because a pulse which is slow in relation to the temperature is almost pathognomonic of typhoid fever. A slow pulse in meningitis of any type, contrary to the impression given by most text-books, is, on the other hand, very unusual and occurs only where there is a considerable increase in cerebral pressure. There is, however, nothing in the story in this case to make us suppose that there is any very marked increase in cerebral pressure. The slow pulse, therefore, is a point in favor of typhoid fever and against meningitis. The respiration is rapid in comparison with the pulse but not in comparison with the temperature; that is to say, the rate of the respiration is what is to be

expected with the fever, and, therefore, does not point to any pulmonary affection.

There has been no change in the physical examination since he came into the hospital. He is, as you see, well-developed and nourished and of good color. It is evident from the way he wrinkles his forehead that he has a headache and he says that this headache is frontal. It is evident, also, from the way in which he tells us what his name is and where he lives, that he is perfectly conscious and clear mentally. His eyes are bright. The pupils are equal and react to both light and accommodation. There is no paralysis of any of the eye muscles. You can see by the way he wrinkles his forehead and blows out the match that there is no spasm or paralysis of any of the facial muscles. His voice is clear and he protrudes his tongue in the median line. His tongue is a little dry and moderately coated. His throat is normal. I can move his head forward, but, as you see, it soon begins to hurt him and I cannot bring his chin down on his chest. Notice that his legs do not move as I bring his head forward; that is, Brudzinski's neck sign is absent. His heart and lungs are normal. His abdomen is slightly sunken. There is no muscular spasm or tenderness. There are no evidences of fluid or masses. The liver and spleen are not palpable. The extremities are normal. There is no spasm or paralysis of either his arms or legs. He can move them all freely and, as you see, there is no disturbance of coördination. His muscular strength is normal. His knee-jerks are normal. The cremasteric reflexes are equal and lively. The epigastric reflex is lively, as is the left abdominal. The right abdominal reflex is, however, diminished. There is no ankle clonus. When I flex his thighs to a right angle with the abdomen and then attempt to extend the legs on the thighs, you see that I cannot bring the right leg further than to an angle of 45° with the thigh, and the left further than to an angle of 50° . As anything above 35° is abnormal, we are justified in saying that he has a slight Kernig's sign on both sides. Babinski's and Oppenheim's reflexes are both negative. There is no disturbance of sensation. There is no enlargement of the peripheral lymph-nodes. There is no eruption on the skin and there are no evidences of any old eruptions. There are no rhagades about the mouth or anus, and his eyes are clear.

The urine is cloudy, amber in color, acid in reaction and of a

specific gravity of 1022. It contains no albumin, sugar, acetone or diacetic acid. The sediment is almost entirely made up of amorphous phosphates, with a few cells, but no casts.

The findings on the physical examination are evidently more consistent with the diagnosis of meningitis than with that of typhoid fever. There are none of the usual signs of typhoid present. The spleen is not enlarged, there are no rose spots, and the abdomen is not distended. The lack of distention of the abdomen is not, of course, of much importance, because distention of the abdomen is not at all a constant symptom of typhoid fever in childhood. The absence of rose spots does not count a great deal against typhoid, because, although rose spots are found very constantly in typhoid fever in childhood, they are not always present, and may be present at one time and not at another. The absence of enlargement of the spleen twelve days after the onset of the symptoms is, however, very strong evidence against typhoid fever, as the spleen is almost invariably enlarged in typhoid fever in childhood. The enlargement is, moreover, relatively greater in children than in adults, and appears early. The only positive signs in the physical examination are the slight stiffness of the neck, the slight Kernig's sign on both sides and the diminution of the abdominal reflex on the right. These signs, while very slight, are, nevertheless, all signs which occur in cerebral disease. The neck may, of course, be held stiffly because of trouble in the throat, and there may be an apparent Kernig's sign because of trouble in the nerves, bones or joints of the legs. The abdominal reflexes are, moreover, not always alike, because of some trouble in the peripheral nerves or in the underlying organs. The combination of these three signs, with the lack of any evidence of local cause for any of them, points very strongly, however, to cerebral disease. If there is a cerebral disease, it is, in all probability, as we have already determined, either tubercular or meningococcal meningitis. There is, of course, nothing about these signs which aids us in any way in determining whether the meningitis is tubercular or meningococcal.

Are there any laboratory tests which will help us? We can, of course, make a white blood count and do a tuberculin test.

What aid may we expect from a white count? Meningococcal meningitis is almost invariably accompanied by a polynuclear hyperleucocytosis. This is usually high, but in mild cases may not be very

large. Tuberculous meningitis, which is a form of miliary tuberculosis, theoretically should show no leucocytosis, because in pure miliary tuberculosis there is supposed to be no leucocytosis. As a matter of fact, a moderate and sometimes even a considerable hyperleucocytosis is found almost as often in tuberculous meningitis as is a normal count. If the leucocyte count is high, therefore, it will be a point in favor of meningococcal meningitis, but will not rule out tuberculous meningitis. If it is normal, it will point very strongly against meningococcal meningitis. If there is a moderate increase, it will be of no assistance. The white count has been done in this case and is 14,200; that is to say, there is a slight leucocytosis, and we are no better off than we were before the count was made.

What assistance may we expect from the Von Pirquet tuberculin test? At least 60 per cent. of the children of the hospital class of this age in Boston will show a positive tuberculin test, because this proportion of the population at this age is infected with tuberculosis. If the test is positive, therefore, the positive reaction does not necessarily mean that he has tuberculous meningitis. It may just as well be due to some small focus of infection somewhere else in the body. The tuberculin test may or may not be positive in tuberculous meningitis. If the system is overwhelmed by the infection in tuberculous meningitis, which is, as I have already said, simply one of the manifestations of acute miliary tuberculosis in which the system is often overwhelmed, the reaction will be negative. If the system is not overwhelmed, the reaction will be positive. It is evident, therefore, that whether the reaction is positive or negative, no conclusions of any value can be drawn from it. The tuberculin test was done when this boy came into the hospital, forty-eight hours ago, and is, and has been, negative. This information, however, is of no practical value to us.

Some of you will want to know, I am sure, whether blood cultures have been made for the presence of typhoid organisms, or the Widal test has been done. Neither of these things has been done. It is too late to expect positive blood cultures, even if this disease is typhoid fever, and the Widal test has been neglected. A positive Widal test at this stage would be almost certain evidence that he has typhoid fever, as at this time, the twelfth day of the disease, it should be positive, if he has typhoid fever. A negative result would be

strongly against typhoid, but would not exclude it. The Widal test would, therefore, be of great assistance, if there was no other more certain way of arriving at a diagnosis.

The quickest, easiest and most certain way to arrive at a diagnosis between meningitis and typhoid fever in this instance, and also to determine what form of meningitis he has, if it is meningitis, is, of course, a lumbar puncture.

A lumbar puncture has been done on this boy. Thirty cubic centimetres of fluid, under slightly increased pressure, were allowed to escape. This fluid, when it escaped, seemed to be clear, but, when it is compared with water, it is, as you see, slightly opalescent. It contains 170 cells to the cubic millimetre. Ninety-eight per cent. of these cells are small mononuclear cells and 2 per cent. are polynuclear neutrophils. The Noguchi test for globulin is positive. The fluid does not reduce Fehling's solution. Careful examination of smears has failed to show any microorganisms.

We are justified, of course, in ruling out typhoid fever on the strength of the findings of the examination of the spinal fluid, which shows positive evidences of cerebral inflammation. These findings are entirely inconsistent with influenzal and pneumococcal meningitis and meningitis due to the pus organisms. They are also inconsistent with a cerebral tumor. There may be an increase in the number of cells in a cerebral tumor and perhaps a positive globulin test, but the fluid will be clear and Fehling's solution will be reduced. They also rule out meningococcal meningitis. It is true that during the convalescent stage of meningococcal meningitis the fluid becomes less and less turbid, the cell formula changes toward the mononuclear and the organisms disappear. This boy is, however, not convalescent. His symptoms are, on the contrary, progressing. The cerebrospinal fluid, therefore, would be purulent or very turbid. The cell formula would be mostly polynuclear and meningococci would be present. Furthermore, Fehling's solution would probably be reduced.

The findings in the fluid correspond exactly to those in tuberculous meningitis. They are, however, not inconsistent with encephalitis, whether primary or as a manifestation of infantile paralysis, and syphilitic meningitis. In none of these are there any demonstrable organisms, and in all of them the fluid is clear or slightly opalescent and the cell formula mononuclear. The Noguchi test may be positive in all of them. Fehling's solution is, however, usually reduced in

these conditions, while it is not in tuberculous meningitis. In the absence of tubercle bacilli in the cerebrospinal fluid, are we justified in excluding these conditions? It seems to me that we are justified in excluding them on the basis of the onset and symptoms, as we have already done, although we cannot do so on the findings in the cerebrospinal fluid.

Are we justified in making the diagnosis of tuberculous meningitis without finding tubercle bacilli in the cerebrospinal fluid? It seems to me that in this instance we are. It is undoubtedly true that tubercle bacilli are present in the cerebrospinal fluid in every case of tuberculous meningitis at some time or other. It is also true that certain experts are able to find tubercle bacilli in almost every instance. The average physician and house-officer very seldom finds them, however, no matter how hard he tries. It is the exception, I regret to say, for the house-officers in the Children's Hospital to find the organisms. I, therefore, feel justified in making the diagnosis of tuberculous meningitis in this instance.

What shall we tell the family as to the diagnosis? It seems to me that the only fair thing to do is to tell them that, although it has not been absolutely proven, everything points to the diagnosis of tuberculous meningitis; that we believe the child has tuberculous meningitis, but are willing to admit there is a possibility that we are mistaken, because we have not found tubercle bacilli. We should emphasize very strongly, however, that in spite of the fact that we have not found the organisms, we are almost as certain that he has the disease as if we had found them. Formerly I used to be willing to make an absolute diagnosis of tuberculous meningitis without reservations in cases of this sort, even when the tubercle bacilli had not been found. I have, however, occasionally been misled, so that I now make a reservation when no tubercle bacilli have been found.

If this boy has tuberculous meningitis, as we believe he has, there is practically no hope for his recovery. It is true that between ten and twenty cases of recovery from tuberculous meningitis have been collected from the literature. In a number of these, however, the diagnosis of tuberculous meningitis seems somewhat doubtful. The only cases, which can be accepted as proven, are those in which tubercle bacilli were certainly found in the cerebrospinal fluid, those in which animals died after inoculation with the cerebrospinal fluid,

and those in which death occurred later, either from tuberculous meningitis or some other disease, and in which the lesions of a former tubercular infection of the meninges were found. Granting that the data in the few cases which are said to have recovered from tuberculous meningitis are correct, the number of cases which have recovered is so small in comparison with the thousands or hundreds of thousands which have died of the disease that we are not justified in considering that in any individual case there is any hope for recovery. Tuberculous meningitis should be looked upon at present as an incurable disease. It is not right, therefore, to give any hope to the parents of a child whom we believe to have tuberculous meningitis, except in so far that we may tell them that, when we have not found tubercle bacilli, it is possible that the diagnosis may be wrong and that on account of this possibility the child may have a chance for recovery. They should certainly be told that if the diagnosis is correct, as we believe, there is no chance for recovery.

It is very difficult to tell how long a child with tuberculous meningitis will live. In general, I am confident that tuberculous meningitis is a disease of shorter duration than would seem from the descriptions in the text-books. We can say, I think, very definitely that babies almost never live over three weeks from the onset, and seldom over two weeks. Children, I feel certain, rarely live over six weeks from the onset of the symptoms, and seldom over four weeks. When children live for months with tuberculous meningitis, I am very positive that there has been or is a mistake in the diagnosis and that the true condition is the chronic type of meningococcal meningitis, or some form of encephalitis. Twelve days have elapsed since the onset of the symptoms in this boy. Except for the headache he has very few symptoms at present and the physical signs are very slight; that is, the progress of the disease has been slow in this instance. I do not dare or care to make a definite prognosis as to how long he will live. Nevertheless, I shall be much surprised if he is alive at our exercise two weeks from to-day.*

You will read much in the text-books about remissions in the symptoms in tuberculous meningitis. These, in my experience, do not occur nearly as frequently as one would suppose from reading the text-books. They do occur sometimes, however, and you must be on

* He died on the twelfth day.

your guard and not be deceived by them, or back down on your diagnosis when it has been made on good grounds. I remember one very striking one in a case of my own in which a child that had been unconscious for a week or more and had been fed with a tube suddenly sat up in bed one morning, recognized his family, asked for his breakfast and ate it. It was very hard for me to convince the parents that the diagnosis and the prognosis were the same that morning that they had been during the past week. Nevertheless, he died that afternoon.

There is no curative treatment for tuberculous meningitis. All that we can do is to endeavor to make the patient more comfortable and to alleviate the symptoms. Fortunately, in general, the symptoms are more distressing to the family than to the patient. As a rule, the patient suffers but little. This boy is suffering far more than is usual in that he retains his consciousness and appreciates that he has a headache. Usually children with tuberculous meningitis do not suffer at all, although it often seems as if they did. They are not conscious during their convulsions, and do not appreciate that they are twitching or rigid, or crying out. Convulsions, twitching and screaming are usually the result of an increase in the quantity of cerebrospinal fluid. They can usually be relieved by the withdrawal of fluid by lumbar puncture, which relieves the pressure. Repeated lumbar punctures are the best treatment for these symptoms. If lumbar punctures are for any reason contra-indicated or do not relieve these symptoms, they should be controlled by chloral, bromide or morphine.

Sooner or later, almost every child with tuberculous meningitis becomes unable to swallow. The question then arises as to whether they should be fed with a tube or not. My own feeling is that they should be fed with a tube, because I know that we are all human, and hence fallible, and that, therefore, we may be mistaken in our diagnosis, even when we are the most certain that we are right. If, however, the parents object to having the child fed with a tube, as they often do, I do not press it. For the same reason, that is, human fallibility, every child with tuberculous meningitis should be treated as if it had a curable condition, and everything should be done for it, as would be done if it had a disease from which it was certain to recover. Nothing should be neglected because we believe that the child cannot recover.

MALNUTRITION IN CHILDREN—A CLASS CLINIC

GIVEN TO FOURTH-YEAR STUDENTS, TUFTS COLLEGE MEDICAL SCHOOL, AND TO MEMBERS
OF THE TRAINING CLASS FOR NUTRITION WORKERS

By WM. R. P. EMERSON, M.D.
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THE METHOD OF SELECTION

By means of a routine weighing and measuring of children sent to the Out-patient Clinic for other than nutritional reasons, we determine which of these children are seven per cent. or more underweight for their height. After long experimentation we have found that the best single rule of selection for malnutrition is based upon the relation existing between weight and height and that *a body weight habitually seven per cent. or more under the average weight for the child's height is not equal to maintaining him in normal health.* There are other types of cases which cannot be gone into at this time, but the seven per cent. rule determines the great majority of those children who are in need of nutritional care.

WEIGHT AND HEIGHT STANDARDS

Table G provides a convenient basis of reference showing the average weight of boys and girls at various heights as well as the weights which are 7 and 10 per cent. under the standard. Extended studies have shown conclusively that at least one-third of the children in this country of school and pre-school ages are sufficiently underweight for their height as to require treatment for malnutrition. One must bear in mind that these standards are low, as they include the measurements of subnormal children (approximately 30 per cent.) as well as of normal and overweight children. However, the proportion of overweight children is small and does not begin to offset those who are underweight.

HOW THE CLASS WAS BROUGHT TOGETHER

Postal cards were sent to the parents of the children who on previous visits to the clinic had been found to come under the 7 per cent. rule. The condition of each child was explained to the parents

and an understanding was reached about the coöperation necessary to insure success.

THE NUTRITION WORKER

A most important factor is the nutrition worker who has charge of the class and acts as general executive assistant to the physician in charge. This position does not require extensive special training but it must have common sense, initiative, reasonable judgment, considerable tact and executive ability as well as accurate training in the essentials of this work.*

HISTORY AND DIAGNOSIS

A very complete history record (J.) is taken of each child and he is given a thorough physical, mental and social examination. The nutrition record is central, but as many specialists are brought in as are required by the needs of the case. *No defect is considered in isolation.* Each examiner receives a copy of the records made by each of the others.

All of this material is entered in the form which you have just seen and on this basis the nutrition diagnosis is made. A very valuable aid in getting the data needed is a record kept for forty-eight consecutive hours of the child's activities and occupations. This includes a statement of the food taken, food and other health habits.

"FREE TO GAIN"

The next step depends upon the nutrition worker for much of its effectiveness. Failure to accomplish it stands in the way of progress in at least half the cases. On the basis of the diagnosis made every phase of the child's life is considered and all medical, home, educational and other social agencies available are brought to bear upon each case.

(1) To remove all obstructions, as diseased tonsils and adenoids, bad food and other faulty health habits, too long school hours, etc., in order that *the child may be made physically "free to gain"*—to use the expression which we have found best describes the condition needed as a foundation for treatment.

(2) To make use of all possible means of appealing to the child's

* The results in this class are due in large part to the nutrition worker in charge—Miss Mabel Skilton, Secretary of Nutrition Clinics for Delicate Children.

imagination in building up a more satisfactory system of interests and habits *following in all instances the line of least resistance.*

CLASS PRELIMINARIES

The room for the class should be large enough to accommodate about fifty people with comfort. The children, twenty in number, are seated in two rows. Back of each child sits his mother and in the rear students and other observers. The nutrition worker and the teacher or school nurse, if the presence of either can be secured, sit in front or at the side where their interest can be evident to the children.

On the wall in front of the children are placed the record charts arranged in two rows, one above the other, and the chart of each child directly before him.

The children come to the classroom at an appointed time accompanied by their parents and are weighed by the nutrition worker, who also inspects their diet books and reckons the number of calories shown in the two-day food record. She takes careful note of possible reasons for the gain or loss of the week just past and makes use of these conferences to secure additional data for the child's history record.

The weight line for the week is then drawn on the record chart; a blue star is given for rest periods, a red star shows regular mid-morning and afternoon lunches—provided in each case none have been missed; a gold star is given to the child who has made the largest gain during the week and to him is given the place of honor at the head of the class. The average number of calories taken each day is shown as well as important events and conditions of the week which have bearing upon progress.

This weighing is really the culmination of the week's work. It should be made an impressive and enjoyable ceremony. For a child to come in after a week's serious effort and have his weighing a mere matter of casual routine throws away one of the chief resources of the work.

CLASS PROCEDURE

If all these preliminaries have been properly attended to before the time of the doctor's coming to the class, it is possible for him to do his part in the class exercise in about half an hour. He takes up

each case in turn, discussing the week's record of the child, making it clear what is the meaning of the evidence and what must be accomplished during the coming week. Little time is spent on those who have gained, but attention is focussed on the few who are standing still or are losing. The parent and the nutrition worker are brought into these discussions and the responsibilities of each as well as those of the child are clearly defined.

Naturally individual cases will require conference and further examination at the close of the class period, but it is surprising how large a part of the work can be accomplished within the class time.

ILLUSTRATIVE MATERIAL

In addition to the forms and records already shown, several tables and charts will help you to understand the work. On Chart A is given what may be called the "Essential Machinery"; on Chart B, the "Essential Ideas" governing the work; and the "Essentials of Health," on Chart C, which we strive to have constantly in the minds of parents, children and workers as the goal to be reached. The statements regarding the "Class Method" (D) have been formulated in order to emphasize this most important agency. Failure to gain cells for further examination is listed in Chart E.

We have here also the weight records of the children which will be referred to in the presentation of the individual cases (M, N, O, P, Q, R, S). Tables F and I show the results of what little statistical work it has seemed worth while to have done with the records accumulated. Chart L is an index of the cases used for reference and as a convenient means of checking up progress without consulting individual records.

INDIVIDUAL CASES

There are fifteen members of this class—eight boys and seven girls—ranging in age from seven to fourteen years and in underweight from 7 to 22 per cent. Three of the girls are excluded from the statement of results as they required treatment in other clinics before they could become "Free to Gain."

For convenience they are considered in four groups, according to the causes of malnutrition:

- I. MEDICAL—4 cases.
- II. SINGLE CAUSE EASILY ADJUSTED—3 cases.
- III. COMPLICATION OF CAUSES, GOOD HOME COÖPERATION—6 cases.
- IV. COMPLICATION OF CAUSES, POOR HOME COÖPERATION—2 cases.

In an hour's time it is not possible to go into detail but I will indicate the essential points in the diagnosis and treatment of each of the four groups.

Group I

Bessie A., and her sister Jennie A., come from a family which illustrates to a marked degree the evil effects upon nutrition of a lack of home control. These children have become artists in having their own way. Jennie rules the home. She is supposed to take after her father and is thin because he is. She likes chocolates because he does. The father sleeps on four pillows, therefore Jennie must have at least three! The parents wish to coöperate and try to do so, but neither of them is efficient.

Bessie's daily program showed certain days on which less than seven minutes were spent in the open air. Both children were up late at night, had too much candy and other sweets, lived in overheated rooms and were constantly overfatigued. The weight charts in both cases show at first no stars for lunch or rest periods, but when the children and their mother saw the other children gaining well, lunches were begun, and later both lunches and rest periods were faithfully attended to.

Their food habits were corrected and both children responded to treatment, each showing a gain of three pounds in two weeks. Jennie continued to gain slowly but Bessie gradually lost in spite of everything done over a period of eight weeks. This loss of weight occurring under circumstances which should have caused a gain, led to a Wassermann test, although there was nothing in the history or the physical examination to indicate specific trouble. The test was positive, and the patient has been turned over to another department for treatment until she becomes "free to gain." The weight chart as a means of diagnosis has great value.

Jennie was taken to the Farm Home and made rapid gain, but on her return she was bowled over by school examinations, neglected her rest periods on account of attending school all day and fell away from the progress she was making. This illustrates the significance of these factors.

Mabel P. Physical examination showed evidence of diseased tonsils and adenoids. Her foster mother would not give consent to

have the necessary operation performed, and the throat department of one of the large hospitals did not consider the operation urgent. The child is troubled with frequent colds. Here again the weight chart is a valuable means of diagnosis. Increased feeding brought about an initial gain of two pounds during the first month, but during the next two months she did not vary half a pound, not being "free to gain." Meanwhile the standards which she should have been meeting have been advancing and she is farther from her expected weight line than she was when she entered the class. A return to normal is not to be expected until the operation has been performed.

Ruth M. had the tea habit. On its correction she made good gain during the first four weeks. She then began to lose weight because of a bronchitis and earache. At the start she was 22 per cent. underweight following an attack of influenza with pneumonia complications. She is the only member of her family who is not well and strong and up to the time of the epidemic she was in good condition. Her failure to gain, together with her bronchitis, have led to a suspicion that the cause of her malnutrition is tuberculosis, and she is now under observation in that department.

Group II

Moses and John M. are brothers. The diagnosis shows a single cause in each case—faulty diet. The mother considered soups especially nourishing and literally fed her family on thin soups. The relegation of this course to its proper place and the substitution of foods of high caloric value caused immediate gain. John gained nearly five pounds in two weeks, going over the expected weight line and graduating in that time. Moses was obliged to leave the class on account of illness in the home, but he was only one pound under the average at that time.

Henry C. was 10 per cent. underweight on account of improper food. In four weeks he gained three pounds and at the close of the term was well started on his way to the goal.

Group III

Marie M. and her mother both fall down on their jobs. She was 13 per cent. underweight and reported by her teacher to be "too tired to learn." The amount of food taken was increased by about

60 per cent. and the school session limited to 10.30, with the result that gain in weight began immediately and continued for three months at the rate of over five ounces a week, 300 per cent. of the expected gain for a girl of nine years. Then came a week in which Marie indulged in excessive jumping of the rope, was overfatigued, unable to take the amount of food needed and lost in weight. All then went forward until loss began because of her being awakened early by a little brother who slept with her. A change to another bed brought over two pounds gain in a single week. Then came pressure on account of backwardness in school work and the full day program was insisted upon with a consequent loss at the rate of nearly a half pound a week.

This case well illustrates the home and school factors which must be controlled before progress can be made.

Marie did not care enough, but Walter B. not only took care but was worried all the time. He is excellent in his school work, but even there he is recognized to be overburdened. The family is poor and the mother is always ill. Her youngest child of seven is fifteen months old and she has had three miscarriages in the past year. Walter is under twelve and 14 per cent. underweight, but he has much of the housework to do and worries about these home cares.

Despite these limitations during the six weeks that Walter was under treatment he gained in weight at the rate of half pound a week. He had enuresis and would not lie down for a rest period for fear of an accident. For the same reason he was unwilling to drink milk.

Group IV

Florence and Mary K. were $8\frac{1}{2}$ and 16 per cent. underweight respectively. The diagnosis in each case was overfatigue. The parents are very coöperative—the father sends letters of appreciation. There are three children in the family, all are underweight and “overgrown,” the father and mother are both underweight. Both girls require long rest periods. Florence has been wearing plates on her teeth, which are uncomfortable and make her nervous. For two weeks new plates made it impossible for her to close her mouth or chew her food, as the teeth did not meet. During this time she barely held her own. Notice by the weight

charts that Mary made nearly ten pounds gain in fifteen weeks and Florence twelve pounds in twenty-three weeks.

James and Fred G. are excellent students. The former was 18 and the latter 9 per cent. underweight. In this instance two members of a family showed almost identical diagnoses. Both had a fear of nausea and were afraid to eat sufficient food. The mother was overanxious but it was not difficult to turn her anxiety into reasonable care. As there was no apparent cause for their frequent vomiting, they were told that nausea belonged to "finicky" children and that sensible boys had no reason for indulging in the habit. Observe in their charts the remarkable gains made.

The occasion for their entering the class was that James had heard me speak at school and he took up with his mother the desirability of entering his brother and himself in a class. Their setbacks occurred the same weeks and were due to the same causes—reaction to grippe, the illness of mother and extra work, James being kept after school for unfinished work, while an interesting plateau appears in Fred's record where he remained at a standstill for two weeks during the time that he was preparing for his "First Communion" at Church.

Tony K. is another case of overanxiety. He cares more for books than for anything "except mother." He would rather read than eat. He worries about his school and his mother who is ill. He worries about his malnutrition and is upset when he fails to make gain. Grippe, a sprained arm, school examinations, abdominal pains (an X-ray revealed no cause) and fainting spells have kept him from progress. When sent to the Farm Home he became homesick and was unable to remain. He is sensitive and moody. He needs sympathy and encouragement, which are difficult to accomplish under the conditions of his life.

Dan C. is the son of a well-known physician. He was admitted to the group because his parents realized that he needed the advantages of the class method. The diagnosis was that he was not ready to try seriously to overcome his 10 per cent. underweight. He was very sure that he could gain when he was ready to try. He has quick perceptions, sees the situation, but postpones action. He had a hurried breakfast because the bus called to take him to school before he had finished eating. It developed in class that he was in the habit

of waking early and reading in bed before breakfast. This caused overfatigue. In other ways he was well guarded, as he attends an open-air school, where he has opportunity for rest periods, lunches and other means of attaining the essentials of health. He has been in the class but a short time, but began to gain at once, and expects soon to reach his normal weight.

SUMMARY

This class had its own individual characteristics but it represents very well the types of problems with which the physician has to deal. Fifteen types of physical defect were found in the examinations averaging 3.25 a child (5.3 for those not "free to gain," see Table F), yet the difficulties which appear in the diagnosis were more than anything else social and an understanding of the means required to meet them is absolutely necessary to successful treatment.

In Table I will be found a comparative statement of the actual and expected weight gains made by the members of the class. It should be examined in connection with the reports already made of the various cases. The class as a whole made 485 per cent. of the expected gain for their ages. The boys made about double the gain made by the girls. Of the twelve children actually under treatment the best record was made by J. M., who gained during the three weeks he was under care at the rate of 1618 per cent. of the expected gain for his age; that is to say, in the time he should be gaining one ounce he was able to bring up his deficiencies in weight at the rate of one pound! The lowest rate in this group was 280 per cent. of the expectation.

Even those who were not yet "free to gain" were able to pass from a condition in which they were constantly falling behind in weight to a gain exceeding expectation. Thus the "specific" case made an average of 250 per cent. and the girls who had obstructed breathing and suspected tuberculosis gained 119 and 114 per cent. respectively.

ESSENTIAL MACHINERY**(Chart A)**

A classroom.

Scales for weekly weighing.

Preliminary two-day record of activities and food.

History record.

Complete physical examination.

Mental examination when necessary.

Complete social examination made by the Nutrition Worker.

Weekly two-day record of food taken.

Weight chart.

ESSENTIAL IDEAS**(Chart B)**

Any child habitually 7 per cent. underweight for his height is malnourished.

The child can be made well in his own home.

Full coöperation of parents is necessary.

Change in food habits, home organization, etc., should be along lines of least resistance.

No defect should be considered in isolation.

Naso-pharyngeal obstructions and other defects interfering with nutrition must be removed in order that the child may be made "free to gain."

Forenoon and afternoon lunches and rest periods are necessary until the child reaches his normal weight.

School and other activities should be limited to prevent overfatigue.

The class method economizes time and gives to each the benefit of the experience of all.

ESSENTIALS OF HEALTH**(Chart C)**

To remove physical, mental and social causes of malnutrition.

To get children to take proper food at frequent intervals.

To prevent over-fatigue.

To secure fresh air by day and by night.

To establish sufficient home control to insure good food and health habits.

THE CLASS METHOD**(Chart D)**

Economizes time of all concerned.

Introduces healthy competition.

Pools experience of all families for the benefit of each.

Favors study and correction of home difficulties by meeting parents under friendly circumstances.

Removes obstacles too great for the authority of the parent and for the undeveloped reason of the child which yield in a surprising manner to interest developed in class.

Removes prejudices and fears through knowledge of results obtained and convinces in a moment when hours spent in argument have failed.

Utilizes the approval of companions as a strong influence in causing a child to do as directed.

FURTHER EXAMINATIONS MADE IN CASE OF FAILURE TO GAIN

(Chart E)

X-ray of Chest, Digestive Tract, etc.
 Special Nose, Throat and Sinus.
 Blood, especially Red Cells and Hemoglobin.
 Wassermann Reaction.
 Temperature Chart Record.
 Skin Tests for Proteins.
 Stools for Parasites, etc.

SUMMARY OF PHYSICAL DEFECTS (F)

	Male.	Female.	Both	Cases not "Free to Gain."	Grand Total
Mouth breather.....	1	1	2	0	2
Tonsils.....	1	0	1	2	3
Deviated septum.....	2	0	2	0	2
Thickened eardrums.....	1	1	2	1	3
Cerumen in ears.....	2	1	3	1	4
Carious teeth.....	4(11)	0	4(11)	2(7)	6(18)
Defective vision.....	1	2	3	1	4
Bronchitis.....	0	0	0	1	1
Adherent prepuce.....	1	0	1	0	1
Tuberculosis.....	0	0	0	1	1
Round shoulders.....	5	2	7	2	9
Spinal curvature.....	1	1	2	0	2
Flatfoot.....	0	0	0	1	1
"Specific".....	0	0	0	1	1
Underweight.....	8	4	12	3	15
Total.....	27	12	39	16	55
Average.....	3.38	3.00	3.25	5.3	3.66
Cases					
With no defects.....	0	0	0	0	0
One defect.....	0	0	0	0	0
Two defects.....	1	1	2	0	2
Three defects.....	2	3	5	0	5
Four defects.....	5	0	5	1	6
Five defects.....	0	0	0	1	0
Six defects.....	0	0	0	1	2
Total.....	8	4	12	3	15

Ages.....	Ten years and under	Over ten years
Average underweight per cent.....	9.9	13.2
Average number defects.....	3.0	4.1

G

Table of average heights and weights of children, also showing weights 7 per cent and 10 per cent under weight for height

BOYS				GIRLS			
Height	Average weight for height	7 % under weight	10 % under weight	Average weight for height	7% under weight	10% under weight	Height
Inches	Pounds	Pounds	Pounds	Pounds	Pounds	Pounds	Inches
35*	32.0	30.0	29.0	31.0	29.0	28.0	35*
36*	33.5	31.0	30.0	32.5	30.0	29.0	36*
37*	34.5	32.0	31.0	33.5	31.0	30.0	37*
38*	36.0	33.5	32.5	35.0	32.5	31.5	38*
39*	37.5	35.0	34.0	36.5	34.0	33.0	39*
40*	39.0	36.5	35.0	38.0	35.5	34.0	40*
41*	40.5	37.5	36.5	39.5	36.5	35.5	41*
42*	42.0	39.0	38.0	41.0	38.0	37.0	42*
43	43.5	40.5	39.0	43.0	40.0	38.5	43
44	45.5	42.5	41.0	44.5	41.5	40.0	44
45	47.5	44.0	43.0	46.5	43.0	42.0	45
46	49.5	46.0	44.5	48.5	45.0	43.5	46
47	51.5	48.0	46.5	51.0	47.5	46.0	47
48	53.5	50.0	48.0	53.5	50.0	48.0	48
49	55.5	51.5	50.0	55.5	51.5	50.0	49
50	59.5	55.0	53.5	58.5	54.5	52.5	50
51	63.0	58.5	56.5	61.0	56.5	55.0	51
52	66.0	61.5	59.5	64.0	59.5	57.5	52
53	69.0	64.0	62.0	67.5	63.0	61.0	53
54	72.5	67.5	65.5	71.0	66.0	64.0	54
55	75.5	70.0	68.0	75.0	70.0	67.5	55
56	79.5	74.0	71.5	78.5	73.0	70.5	56
57	83.5	77.5	75.0	82.0	77.0	74.5	57
58	87.5	81.5	79.0	87.0	81.0	78.5	58
59	91.5	85.0	82.5	91.5	85.0	82.5	59
60	95.0	88.5	85.5	96.5	89.5	87.0	60
61	99.5	92.5	89.5	102.5	95.5	92.5	61
62	105.0	97.5	94.5	110.5	103.0	99.5	62
63	109.5	102.0	98.5	116.0	108.0	104.5	63
64	116.0	108.0	104.5				
65	119.5	111.0	107.5				
66	126.0	117.0	113.5				
67	134.0	124.5	120.5				
68	138.5	129.0	124.5				

The figures for the younger children are taken from "Holt's Diseases of Infancy and Childhood," for the ages from six years on, from the studies of Boas, Burk and Smedley. These latter heights and weights are with indoor clothes but without shoes. In all cases the weights have been approximated to the half-pound.

*Without clothes

H
TABLE SHOWING AVERAGE INCREASES IN WEIGHT AT VARIOUS AGES BY YEARS,
QUARTERS AND WEEKS
Boys

Age.	Year 52 weeks		Quarter 13 weeks		Week Ounces
	Lbs.	Ounces.	Lbs.	ounces.	
6½ to 7½.....	4.3	68.8	1.075	17.2	1.32
8½.....	5.0	80.0	1.25	20.0	1.54
9½.....	5.1	81.6	1.275	20.4	1.57
10½.....	5.8	92.8	1.45	23.2	1.79
11½.....	5.3	84.8	1.325	21.2	1.63
12½.....	6.2	99.2	1.55	24.8	1.91
13½.....	7.9	126.4	1.975	31.6	2.43
14½.....	10.4	166.4	2.6	41.6	3.20
15½.....	12.2	195.2	3.05	48.8	3.75
16½.....	13.6	217.6	3.40	54.4	4.18

Girls

6½ to 7½.....	4.3	68.8	1.075	17.2	1.32
8½.....	4.8	76.8	1.2	19.2	1.47
9½.....	4.9	78.4	1.225	19.6	1.51
10½.....	5.5	88.0	1.375	22.0	1.69
11½.....	6.6	105.6	1.65	26.4	2.03
12½.....	9.2	147.2	2.3	36.8	2.83
13½.....	10.0	160.0	2.5	40.0	3.08
14½.....	9.6	153.6	2.4	38.4	2.95
15½.....	8.4	134.4	2.1	33.6	2.59
16½.....	5.6	89.6	1.4	22.4	1.72
17½.....	3.1	49.6	.775	12.4	.95

TABLE I. COMPARISON OF ACTUAL AND EXPECTED WEIGHT GAINS

Name	Age at Entrance	Weeks in Attendance	Per cent. Under Weight	Gain per Week		Per cent. Actual of Expected
				Actual	Expected	
<i>Boys</i>						
J. M.	9-0	3	8	Ounces 25.3	Ounces 1.57	1618
J. G.	11-5	13	18	14.2	1.63	871
M. M.	7-0	4	7	9.0	1.32	680
F. G.	8-6	13	9	8.0	1.54	520
W. B.	11-8	6	14	8.0	1.91	420
H. C.	12-6	6	10	7.3	2.43	300
D. C.	11-0	5	10	4.8	1.79	270
T. K.	10-11	13	7	4.3	1.79	240
Total..				80.9	13.98	573
<i>Girls</i>						
M. K.	10-3	13	11	9.6	1.69	565
F. K.	13-7	13	6	8.32	2.95	282
M. M.	8-8	13	13	5.28	1.51	350
J. A.	10-8	13	8	4.32	2.03	213
Total..				27.52	8.18	336
Total Boys and Girls.....				108.42	22.16	485

NOTE: Three girls (B. A., R. M. and M. P.) are excluded because they were transferred to other divisions of the Clinic in order that they might be made "free to gain." The records that were kept of their weights show that they gained during the quarter 250, 114 and 119 per cent., respectively, of their expected gain during the period.

FORM FOR HISTORY AND PHYSICAL EXAMINATION

[illegible]

INTERNATIONAL CLINICS

[illegible]

Supplementary Notes:

K

WEIGHT RECORD

Age Years Months
 Height Inches
 Weight Pounds

Name

Average Weight for Height
 Underweight Pounds
 Underweight Pounds
 Per Cent

Weight Date	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
Pounds																										
Underweight																										
Per Cent																										

In proportion to his weight. (Boys, column 2; Girls, column 3). For example, if your son weighs 50 pounds, the bottom line of the square above "50" would be the number of pounds to be placed on the bottom line of the square above "50". The square above 100, 50 and so on up until you reach the figure which corresponds to the normal weight for his height.

Weight the child each week on the same day and at the same hour, as there is some variation in weight at different times of the day. Fill in the weighing at the end of the first week falls on August 5, the second one would be August 15, and so on across the chart. Place a dot in the square corresponding to the date

your son's weight. If your son weighs 70 pounds, in writing the chart, 25 would be the number of pounds to be placed on the bottom line of the square above "70" or the lower left hand side. His expected weight then would then be 70 pounds above that in the vertical column for the first weighing.

(*) An underweight boy of eleven should gain at least 4 pounds in half a year, so that his expected weight then should run from the bottom line of the square indicating 45 pounds to the square indicating 67 pounds at the extreme right. Use a long ruler to connect these two points and thus construct his expected weight line.

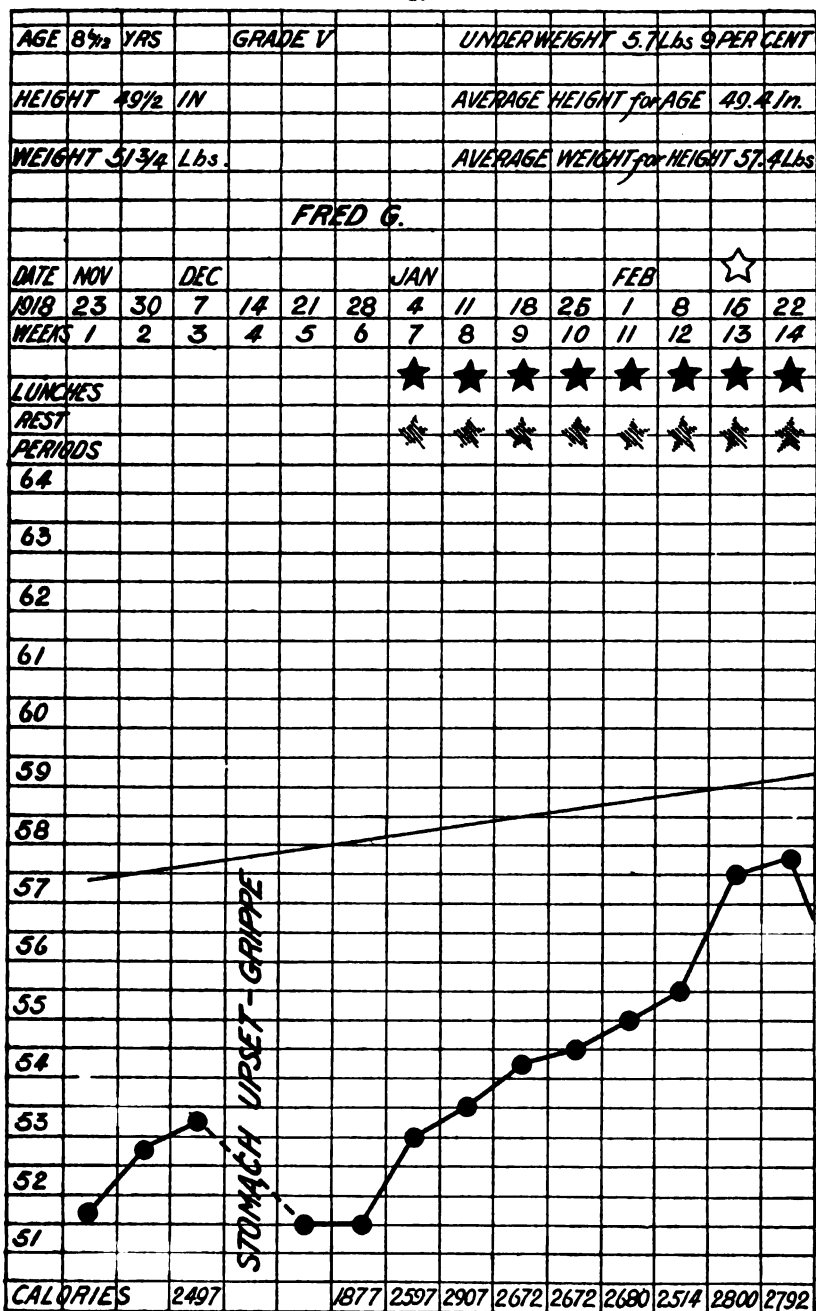
Weight	26	25	24	23	22	21	20	19	18	17	16	15	14	13	12	11	10	9	8	7	6	5	4	3	2	1
Underweight	180.0	170.0	160.0	150.0	140.0	130.0	120.0	110.0	100.0	90.0	80.0	70.0	60.0	50.0	40.0	30.0	20.0	10.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Per Cent	100.0	90.0	80.0	70.0	60.0	50.0	40.0	30.0	20.0	10.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

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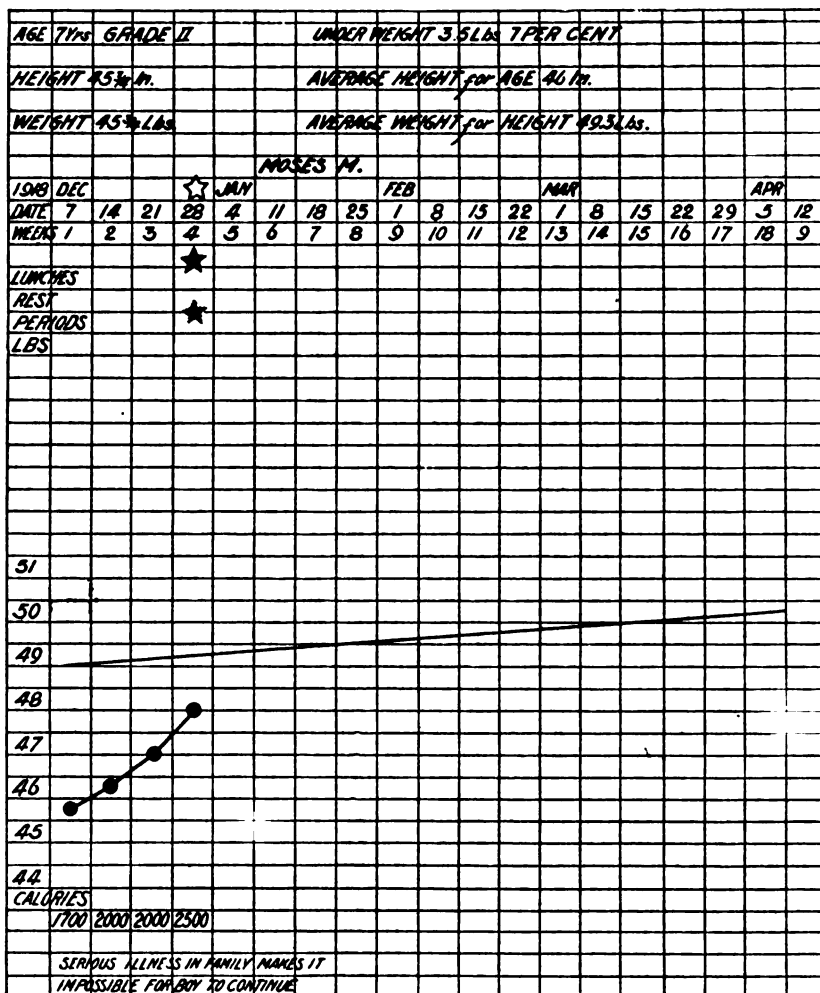
RED STAR Means Daily Lunch
 BLUE STAR Means Daily Rest Periods
 (These may be made with colored crayons.)

Record of Physical Defects		Natalum Clinic The Berkeley Infirmary 1916		Age at Entrance	
Mr. Marie M.	3	1	Adenoids	3	2
Ms. James G.	3	2	Hypertrophic pharyngitis	3	2
Mr. Fred G.	3	2	Tonsils	3	2
Ms. John M.	4	4	Deviated Septum	3	2
Ms. Joseph M.	4	4	Spur	3	2
Ms. Joseph M.	4	4	Open Mouth	3	2
Ms. Joseph M.	4	4	Enlarged cervical glands	3	2
Ms. Joseph M.	4	4	Otitis media-chronic	3	2
Ms. Joseph M.	4	4	Ears thickened	3	2
Ms. Joseph M.	4	4	Bronchitis	3	2
Ms. Joseph M.	4	4	Carious Teeth	3	2
Ms. Joseph M.	4	4	Alveolar Abscess	3	2
Ms. Joseph M.	4	4	Gingivitis	3	2
Ms. Joseph M.	4	4	Cerumen	3	2
Ms. Joseph M.	4	4	Strabismus	3	2
Ms. Joseph M.	4	4	Defective Vision	3	2
Ms. Joseph M.	4	4	Conjunctivitis	3	2
Ms. Joseph M.	4	4	Valvular Heart Disease	3	2
Ms. Joseph M.	4	4	Lateral Curvature	3	2
Ms. Joseph M.	4	4	Round Shoulders	3	2
Ms. Joseph M.	4	4	Enteroptosis	3	2
Ms. Joseph M.	4	4	Adherent Prepuce	3	2
Ms. Joseph M.	4	4	Vaginitis-simple	3	2
Ms. Joseph M.	4	4	Vaginitis-gonorrheal	3	2
Ms. Joseph M.	4	4	Syphilis-hereditary	3	2
Ms. Joseph M.	4	4	Enuresis	3	2
Ms. Joseph M.	4	4	Pin Worms	3	2
Ms. Joseph M.	4	4	Acanthosis	3	2
Ms. Joseph M.	4	4	Pediculosis	3	2
Ms. Joseph M.	4	4	Flat Foot	3	2
Ms. Joseph M.	4	4	Underweight	3	2
Ms. Joseph M.	4	4	Tuberculosis	3	2
Ms. Joseph M.	4	4	Habit Spasm	3	2
Ms. Joseph M.	4	4	Mental Retardation-one year	3	2
Ms. Joseph M.	4	4	Mental Deficiency	3	2
Ms. Joseph M.	4	4	Specific	3	2

N

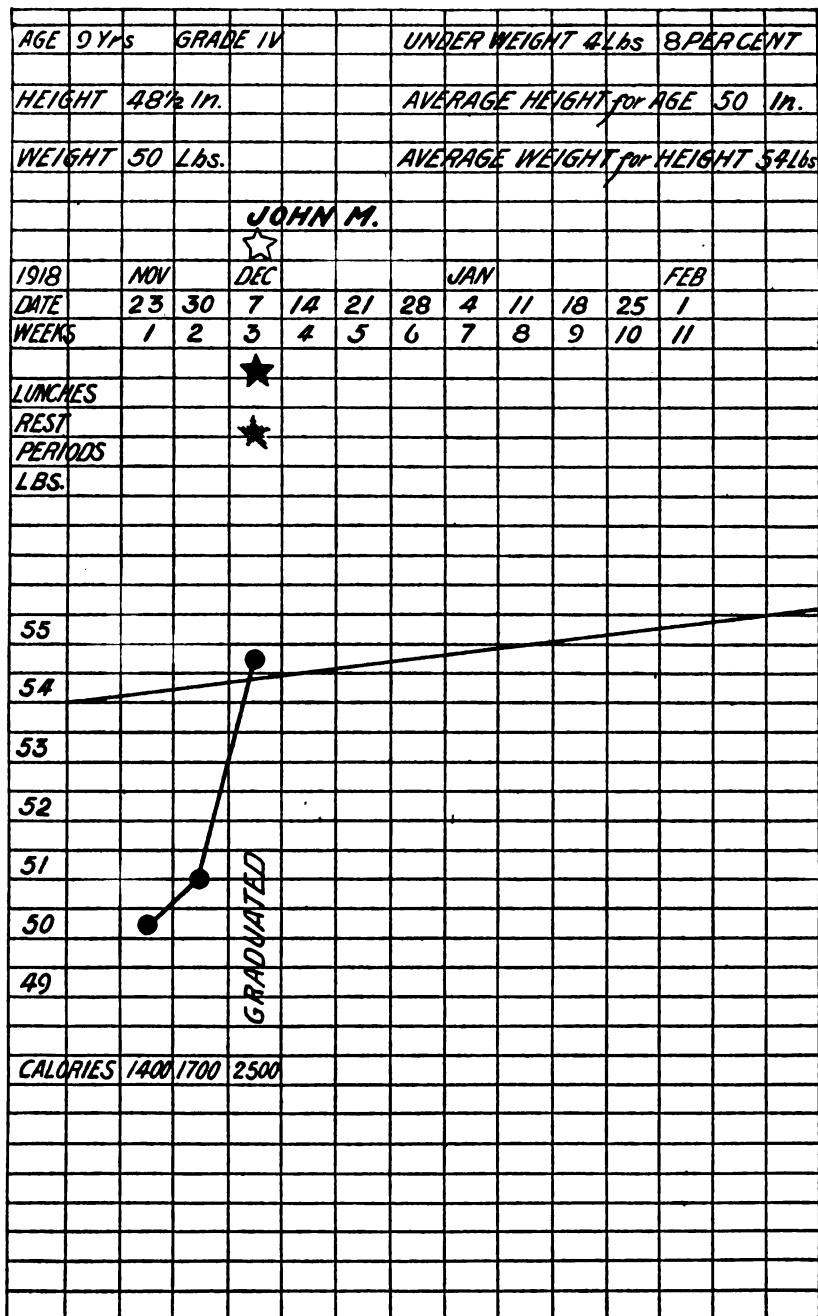


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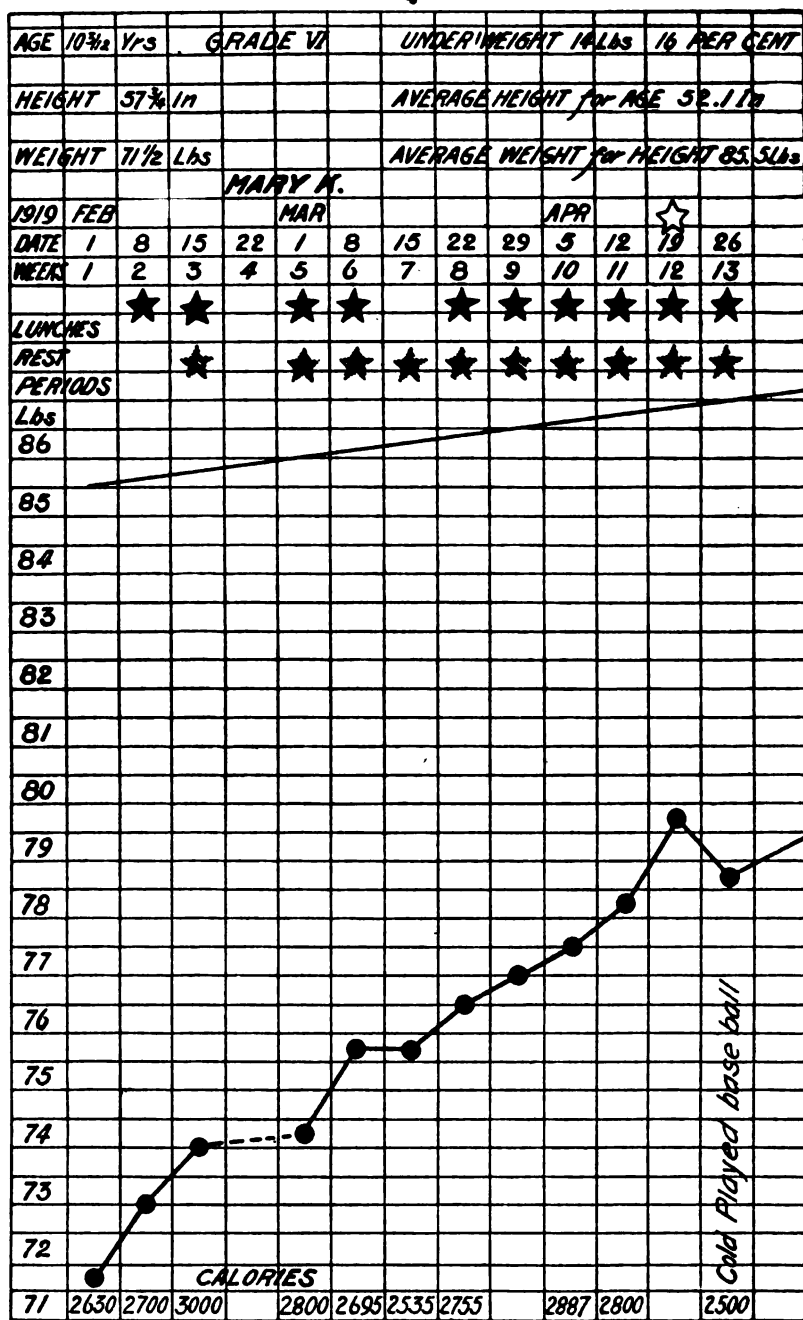
★ GOLD ★ RED ★ BLUE

P

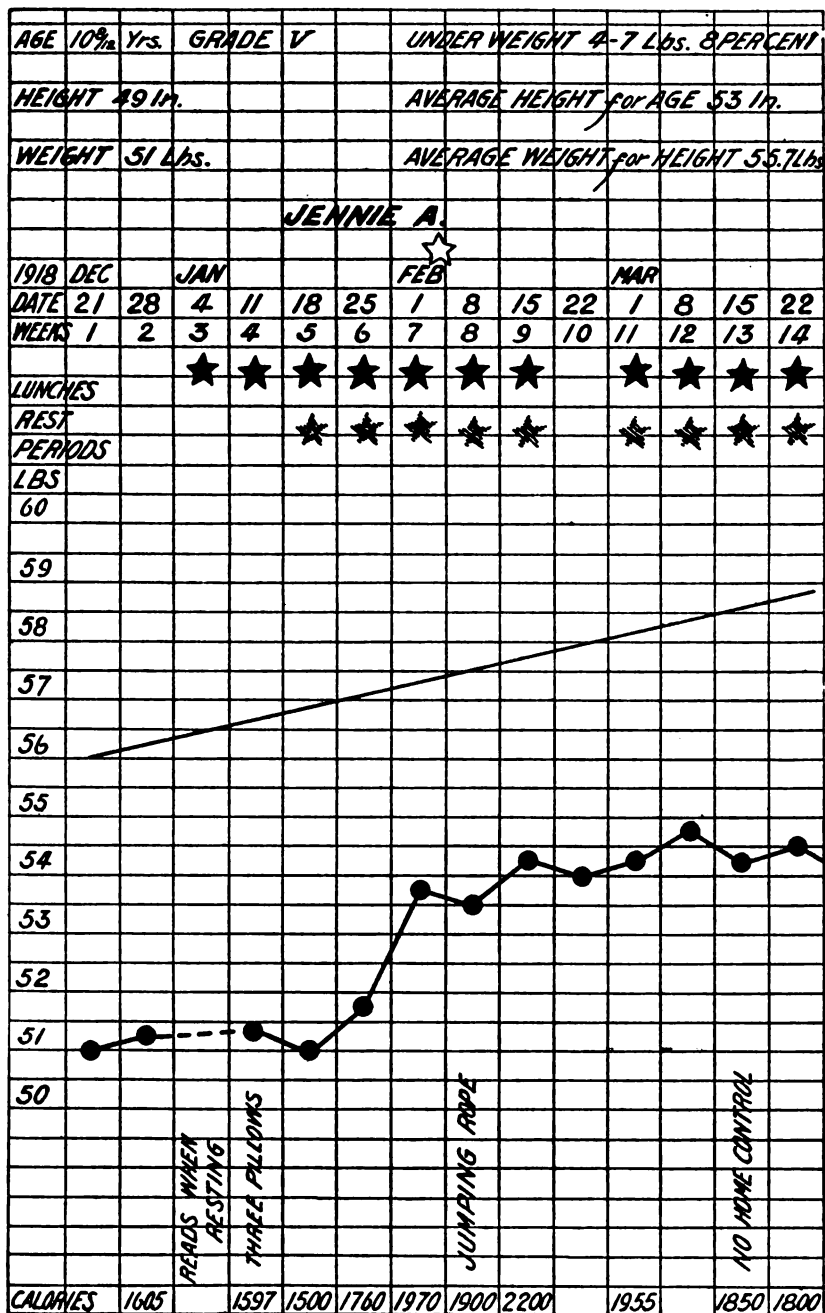


☆ GOLD ★ RED ★ BLUE

Q



T



☆ **GOLD** ☆ **RED** ☆ **BLUE**

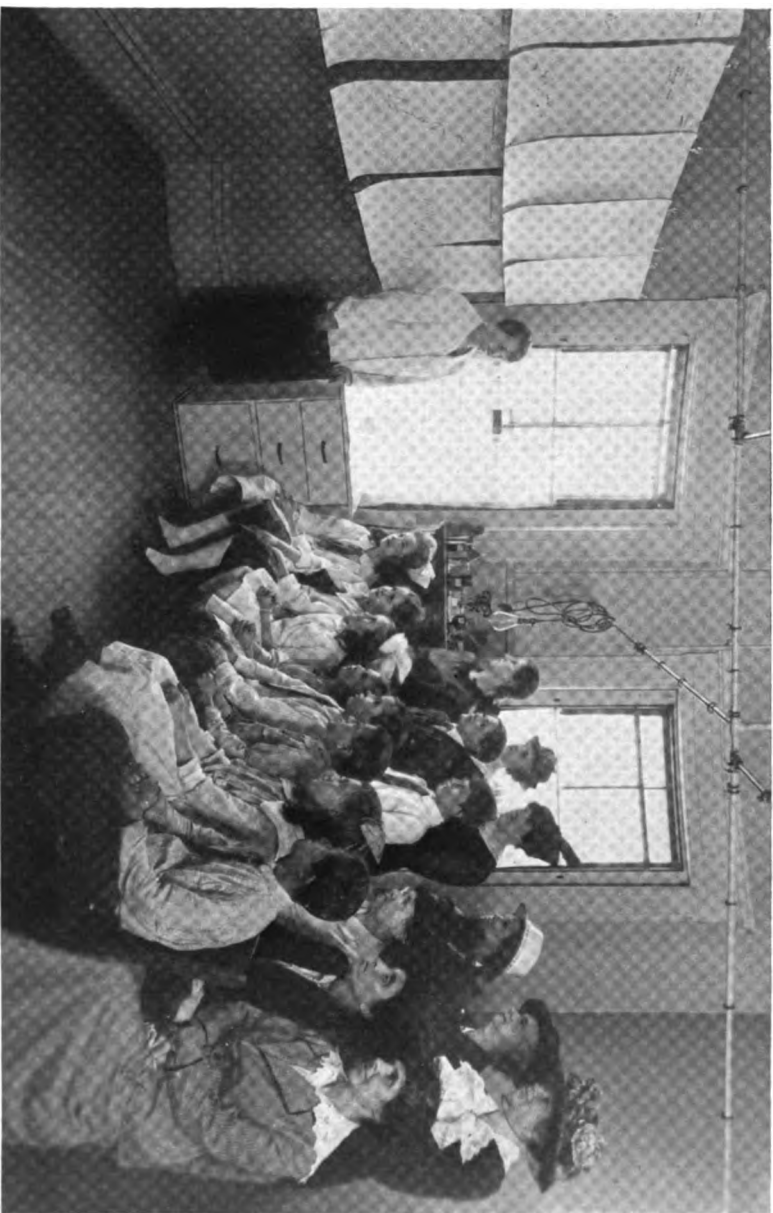
OUTLINE

I. *Report of Class:*

1. The method of selection.
2. Weight and Height Standards.
3. How the Class was Brought Together.
4. The Nutrition Worker.
5. History and Diagnosis.
6. "Free to Gain."
7. Class Preliminaries.
8. Class Procedure.
9. Illustrative Material.
10. Individual Cases.
 - (A) Group I—Medical cases.
 - (B) Group II—Single cause easily adjusted.
 - (C) Group III—Complication of causes, lacking coöperation.
 - (D) Group IV—Complication of causes, good coöperation.

II. *Illustrative Material:*

1. Charts:
 - (A) Essential Machinery (A).
 - (B) Essential Ideas (B).
 - (C) Essentials of Health (C).
 - (D) The Class Method (D).
 - (E) Further Tests to be Used in Case of Failure to Gain (E).
2. Tables:
 - (A) Summary of Physical Defects (F).
 - (B) Weight and Height Standards (G).
 - (C) Expected Weight Increases (H).
 - (D) Comparison of Actual and Expected Weight Gains (I).
3. Forms:
 - (A) Physical Examination and History Record (J).
 - (B) Weight Record (K).
 - (C) Record of Physical Defects (L).
 - (D) Weight Records of Seven Members of Class (M-N-O-P-Q-R-S-T).
4. Photograph of Eleven Members of Class and their Mothers (U).



Nutrition Class Meeting of May 31, 1919, at Berkeley Infirmary. Eleven members present with Florence K. at the head of the Class having gained $2\frac{3}{4}$ pounds in a week's time. The mothers of all the children were also present. The children are of Irish, Hebrew, Armenian, Portuguese and American stock. They represent the various economic needs found in a clinic except that one child comes from a wealthy family which found this opportunity to bring their child into close relations the best means of bringing him up to normal weight.

Surgery

ACUTE ILEAL INTUSSUSCEPTION—ACUTE APPENDICITIS—INTESTINAL OBSTRUCTION DUE TO A MECKEL'S DIVERTICULUM—IN CHILDREN

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THE following three cases, all acute abdominal emergencies, came into our service within three hours of one another and were all operated on during the same evening. The conditions present were all different and yet presented many symptoms in common. The differential diagnosis in the three cases presents a number of points of interest.

The first case, a young girl, Ruth M., five years old, entered Ward 30 of the Cook County Hospital, on the 8th of July, at 6.15 P.M. She came in with the examining room diagnosis of acute appendicitis. The hospital history runs as follows:

"The onset was Friday, July 5th, with acute cramp-like pains in the abdomen. The pains first came on about ten in the morning. There was vomiting of a yellowish and then a greenish material, following the onset of the pain. Some pickles which she had previously eaten were vomited. The mother thinks the girl had some fever also, shortly after the onset of her cramps.

"The pain has been persistently cramp-like, perhaps more severe in the lower abdomen than in the upper, but not more specifically localized. The pain was somewhat relieved following the initial vomiting, but it persisted pretty steadily all day Friday. The girl slept Friday night. Since Saturday she has been listless, almost stupid at times, and has been kept in bed since the onset. The cramp-like pains continued at intervals and were still present when the patient entered the hospital.

"The nausea and vomiting which the patient had Friday morning lasted only about five minutes. There was another attack of vomiting in the afternoon. The next day, Saturday, there was vomiting at 8 in the morning and again in the afternoon. Sunday she vomited similarly and to-day (Monday) she has vomited also, just how many times we did not elicit. Whereas the vomitus at first was greenish in color, the mother states that since then it has been black once or twice, perhaps due to the medicine given by the doctor. Following the taking of milk the vomitus was curdy, and in general, it has consisted only of stomach contents or a little greenish bile. No feculent material has been vomited.

"She has urinated rather frequently since the onset of pain, but urination has not been painful or accompanied by blood or any visible abnormality of the urine. There is no history of previous gastric disturbance, no injury or cough. Her previous illnesses appear to have no bearing on the present trouble. She had anterior poliomyelitis in January, 1916. She was left with a paralysis of the legs, more marked in the right than in the left. The chief involvement at present is in the left foot. She has had no serious other previous illnesses and no operations.

"*Physical examination of scalp, eyes, ears, nose, mouth, pharynx, heart, and lungs are negative. Tonsils are a trifle large and cervical glands barely palpable. On inspection the abdomen seems to be tense and acutely ballooned out. It is markedly tympanitic on percussion. The abdominal wall is a little tense but rigidity is not marked. Tenderness is present and perhaps a little more on the right side of the abdomen than on the left, but not localized to any specific spot and is not marked. The child, when asked, points to the umbilicus as the site of the pain. Auscultation reveals no audible peristaltic sounds. In the midline of the abdomen is a rather vague sausage-shaped tumor mass just below the umbilicus which cannot be palpated per rectum. The examining finger in the rectum comes out stained with blood. The patient's temperature on admission is 100° per rectum, pulse 128, respirations 24. The white cell count of the blood is 13,000. The urine is straw-colored, specific gravity 1026, acid reaction; no albumen or sugar, no cellular elements except an occasional leucocyte.*"

COMMENTS

This is not a case of acute appendicitis at all but a typical case of *intussusception*. The doctor in the examining room in diagnosing acute appendicitis has merely failed to note the mutual relations of the abdominal pain, tenderness, rigidity, vomiting, fever, leucocytosis and abdominal tumor mass. Many of the cases of acute intussusception which I see are similarly diagnosed acute appendicitis by some doctor before they come to the hospital.

This patient's pain was generalized at the start and it has remained generalized. It was cramp-like at the start and it has remained cramp-like. The vomiting which followed the onset has gradually increased in amount and frequency. Remember that the pain and tenderness of acute appendicitis are first general and then become localized to the right lower quadrant, then becoming generalized again only in case rupture occurs and peritonitis develops. The vomiting of appendicitis is first the "reflex" vomiting at the onset and then, later, the vomiting of peritonitis, either general or localized, after rupture of the appendix has occurred.

The patient's abdominal tenderness and rigidity are less marked than we should expect in acute appendicitis. Furthermore, we have here a *movable mid-line tumor* which is not very tender and is sausage-shaped instead of the round or oval boggy mass which an acute appendicitis may rarely produce in this region, when walled off by omentum, but not otherwise adherent to its surroundings.

Lastly, we have blood in clots passed per rectum—a very great rarity in acute appendicitis unless following operation, and then due most often to bleeding from an inverted but unligated appendix stump.

Perhaps the presence of fever and leucocytosis may have misled the examining doctor. They should not have done so because they are relatively constant in ileus which has lasted a few days. In an ileus of four days' duration a moderate leucocytosis is fully as constant a finding as in acute appendicitis!

Perhaps the doctor failed to find out about the bowel movements. The three enemas daily may have been deceptive. Did he ask about the results? If he had, he would have found that little results were obtained from the enemas after the first day; but the frequency of the enemas was in itself suggestive. Why? Because, if the mother

had obtained results, she would not have given three enemas daily, but only one!

It is rather surprising that I cannot feel this tumor mass from the rectum if it be lying in the upper sigmoid, as I should expect, with an ileo-cæcal intussusception of four days' duration. Perhaps this is the exceptional case, not the common ileo-cæcal intussusception but one of the small bowel, the lower ileum. If this be the case, it will explain why blood did not appear in the enemas till the fourth day, instead of on the second or third day, as is more common in acute intussusception of the ileo-cæcal type.

OPERATION

Since we are certain that this is an intussusception and not an acute appendicitis we shall make a midline incision below the navel.

Right para-median incision made. On opening the abdomen the previously palpated mass is felt, but not seen, lying close against the posterior abdominal wall.

We now retract the intestine, bringing this mass into view. It is an ileal intussusception. So much of the mesentery has been invaginated that the mass lies close to the root of the mesentery posteriorly. . . . I am now milking out the intussusceptum and it is a difficult task, because I have to work in the depths of the abdomen. The invagination of the mesentery makes it impossible to bring the mass outside of the belly, until the intussusceptum is released. . . . Now at last I have it freed. See how black and infiltrated with blood the invaginated bowel is. These three or four gray necrotic spots look as if they were on the verge of perforation. The nutrition of the entire intussusceptum is evidently much impaired. I believe it will be safer to resect this entire black hemorrhage bowel rather than to attempt to sew over these gray areas and replace the loop. (Resection of about 14 inches of black bowel was performed and a side-to-side anastomosis by suture was made.)

My anastomosis has been carried out entirely outside the abdomen, well surrounded with towels and pads, and I believe the site of operation is clean enough to justify me in returning the anastomosed bowel to the abdomen without drainage. Whatever infection may be left on the surface of the bowel, I believe will be taken care of by the peritoneum. (Primary closure was accordingly carried out.)

POSTOPERATIVE

The patient was put up in Fowler's position and was given the usual 5 per cent. glucose per rectum, and also hypodermoclysis. She had some fever for several days and finally developed a stitch abscess in the lower angle of the wound. Otherwise recovery was uneventful. The patient left the hospital on August 2 with the wound completely healed. She had had no temperature above 99° for three days before, but she had had a single temperature of 101.4° on July 27. The patient has not again reported since leaving the hospital.

The second patient, a nine-year-old girl, Martha B., entered Ward 30 of the Cook County Hospital on the same evening, July 8, at 8.30 P.M. The examining room diagnosis was *acute appendicitis* and mitral regurgitation.

The patient's history is as follows:

"Last Friday, July 5, three days ago, the patient was first taken ill with headache, which was severe and throbbing in character and most marked over the forehead. The child states that red pimples appeared on her face, which itched and disappeared yesterday after two days' duration. The child slept well on the night of onset, Friday, July 5. The headache, however, continued on Saturday. She felt pretty well on Sunday, July 7.

"This morning, however, Monday, July 8, she had what her mother called a 'sneezing spell,' but without cough or sore throat, and she complained of headache. Abdominal pain was noted for the first time this morning. It was cramp-like, localized in the right lower quadrant of the abdomen, and did not radiate either to the back or to the front or below.

"Vomiting occurred a short time after the onset of the pain in the abdomen. The vomitus was yellow and contained food material, but no visible blood. Vomiting was of about five minutes' duration and has not been repeated since then. The patient has had a normal bowel movement daily. There have been no chills. There is no history of urinary, respiratory, or other gastro-intestinal disturbance. The child is, however, subject to rather frequent attacks of dizziness. Previous illnesses are: scarlatina, measles, and acute rheumatic fever four years ago. Family history is negative.

"*Physical examination* is as follows:

"Patient is a well-nourished little girl in no severe distress or pain. She enters the hospital with a pulse-rate of 136, temperature of 102.2° per rectum, respirations 28. Her white count, made immediately on entrance, showed 12,800 cells. Urinary examination shows specific gravity 1016, clear, straw-color, acid reaction, no albumen, blood casts or cells.

"Examination of the head, mouth, throat and neck is negative, except for somewhat enlarged tonsils, the right tonsil showing a little follicular exudate. The cervical glands are also barely palpable, a little more perhaps on the right side than on the left and more posteriorly than anteriorly.

"Lungs are negative. Heart: apex beat in the fourth left interspace just outside the mamillary line. Percussion shows the left border about a finger's breadth outside the left mamillary line, and the right border at the right sternal margin. On auscultation there is a well-marked blowing murmur heard over the entire precordium. This is heard best at the apex, but is transmitted upward and also to the left and posteriorly. The second pulmonic sound is somewhat louder than the second aortic. There is only slight heaving to be felt over the lower sternum. There is no evident cyanosis.

"The abdomen is retracted rather than distended. There is some rigidity and resistance of the abdominal muscles, perhaps more on the right side than on the left. Tenderness is present only in the right lower quadrant. There is no palpable mass anywhere. Peristaltic sounds are heard to about normal extent and have about the normal tone quality. Liver, spleen, kidneys are not palpable and do not appear enlarged to percussion. Examination is otherwise negative. Diagnosis of Doctor Lewin in the ward is: acute appendicitis, mitral regurgitation, and follicular tonsillitis."

DISCUSSION

In this case both the doctor in the examining room and Doctor Levin in the ward are agreed on the diagnosis of acute appendicitis; also on the diagnosis of a mitral regurgitation. I believe that they are both correct. This seems to me a typical case of acute appendicitis, although the history at first blush may be a little misleading. Her appendicitis attack began this morning. The complaints which she had previously, namely, her headache and the eruption on the

forehead, may have been the result of a gastro-intestinal upset, but do not have anything to do with her appendicitis in a direct way. This morning she had the typical onset of acute appendicitis with cramp-like pains, followed by vomiting and then by rapidly localizing tenderness and muscle resistance in the right lower quadrant of the abdomen. Leucocytosis and fever complete the picture.

The absence of abnormal distention, the normal daily bowel movements, the presence of normal peristaltic sounds, normal both in quantity and tone quality eliminate the possibility of an ileus, either acute or chronic. The normal urine and the absence of loin tenderness eliminates the possibility of a right-sided pyelitis, or renal or ureteral calculus, pyelitis being not such a rare condition in young girls of the poorer classes.

What is the etiology of her appendiceal attack? Is it the result of an acute gastro-enteritis, such as her headache and furred tongue might possibly indicate, or is it in some way connected with her old follicular and chronic endocarditis, as perhaps Rosenow might insist? It seems to me that it is extremely difficult to draw any conclusion along this line in the present case. Even if streptococci were found, both in the ileum and wall of the appendix as well as in the tonsillar crypts, I still should not be convinced that a connection between the two was indisputable.

What has her mitral regurgitation to do with the indications for operation in this case? I consider it an added indication rather than a contra-indication. If her mitral regurgitation should contra-indicate operation for an as yet unruptured appendix, it should still more strongly contra-indicate her passing through an attack of peritonitis and abscess formation, which would be a much greater strain on it. She should by all means be operated upon as promptly as possible. And since her heart lesion is only mild in degree and perfectly compensated, I do not anticipate that she will have the least difficulty with her heart during operation or convalescence.

OPERATION

The peritoneal cavity is opened through a right rectus incision. The head of the cæcum and the appendix are brought up into the wound. The appendix is found swollen, acutely inflamed, and covered with a fibrinous exudate. The mesenterium is ligated with catgut.

A linen purse-string is inserted around the base of the appendix, the appendix cut off and the stump tied and inverted inside the purse-string suture after sterilization of the mucosa of the stump with carbolic acid and alcohol. The wound is closed in layers without drainage.

POSTOPERATIVE

The patient made an uneventful recovery. The wound healed by primary intention. The patient suffered from gas pains only during the first day after operation. The patient left the hospital on July 27, 19 days after she entered. The stitches were out, the wound was completely closed, the patient was feeling well in every way, having been entirely free from temperature for three days before leaving.

The third patient, Mary R., a school girl, aged seven, entered Ward 30 on the same evening, July 8, at 8.50 P.M. The examining diagnosis—shall I say as usual in cases of right-sided abdominal pain—was “suspected acute appendicitis.”

The history reads as follows:

“The onset of the patient’s present illness was yesterday afternoon at 2 o’clock, July 7. Abdominal pain was the first symptom noted. It was cramp-like, most marked about the region of the umbilicus, and radiated to the back and also to the symphysis pubis. The initial pain recurred and was so severe that the child screamed as the paroxysms of cramps returned. They return every two to five minutes as she is observed in the ward and, according to the mother’s story, have done so ever since the onset and have remained approximately of the same character and intensity throughout the course.

“The patient vomited just after the onset of her pain yesterday afternoon. The vomitus consisted of her dinner. She has vomited frequently, at least twenty times since then. The vomitus has been yellowish, but has not contained blood. There has been no foul odor. The patient had a severe chill at about the time the initial pain started. It was so severe that her teeth chattered and she shook all over; then broke out with a cold sweat. This chill has been repeated several times to-day and was also present yesterday.

“The patient has been constipated for the last two days. There

has been no bowel movement at all in this time. Previous to the present illness her bowel movements were regular daily.

"The mother denies any disturbances of the urinary, vascular, or respiratory systems or of the gastro-intestinal tract preceding this attack.

"Her single previous illness appears to have no bearing on the present complaint: bronchitis at one and a half years of age.

"*Physical Examination.*—The patient, a well-nourished little girl, is suffering with frequent paroxysms of intense abdominal pain. She entered the hospital with a temperature of 99.2°, pulse 128, respirations 32. A white count made immediately on entrance showed 29,600 white cells. The urine has a specific gravity of 1012, is clear, acid in reaction, and shows no albumen, blood-cells, or casts.

"Examination of the scalp, eyes, ears, nose and throat is negative, except that the tongue is found coated and both tonsils large and hyperæmic. The neck is negative, except for slight bilateral superficial adenopathy.

"The chest appears negative to external examination. The lungs appear normal. The heart is normal, apex beat lying in the fifth interspace inside the mamillary line; no murmurs, no irregularity.

"The abdomen is tensely distended, tympanitic throughout, more in front than in the sides. There is some rigidity present, which appears most marked in the right lower quadrant. Tenderness is also present, chiefly in the right lower quadrant. There is heard an occasional gurgle of peristalsis, rather high pitched in character to accord with the tympany present, but scarcely loud enough to be called a well-marked borborygmus. There are no splashing sounds on palpation. The liver and spleen are not enlarged to palpation or percussion. The kidneys are not palpable. There are no palpable masses in the abdomen. Rectal examination is negative. No mass is felt inside the pelvis. No abnormal resistance is present and no blood appears on the examining finger. The reflexes are all normal."

COMMENTS

What is the matter with this little patient? Has she an appendicitis as the examining room doctor suspects? Certain facts in the history and examination point in that direction, namely, the sudden onset with cramp-like pains around the umbilicus, with pain and

tenderness, later tending to localize in the right lower quadrant, an onset with vomiting, low fever and leucocytosis. On the other hand, there are some symptoms which do not fit in with an acute appendicitis. For instance, the patient's abdomen is unusually tympanitic and distended for an appendix which has not yet ruptured. The severity and persistence of these excruciating paroxysmal cramps is a most unusual symptom in early acute appendicitis, but it is sometimes seen in the later stages when an intestinal obstruction is developing following an abscess or extensive adhesions. The tinkling high-pitched character of the peristaltic sounds indicates dilated loops of bowel, but that peristalsis is present at all tells me that this cannot be an acute complete obstruction. If there is obstruction here, as seems very likely, it must be a chronic obstruction which has rather suddenly become almost, but not quite, complete. When an incomplete obstruction finally becomes complete, peristaltic sounds entirely disappear. The painful cramps likewise disappear, as vigorous hyper-peristalsis gradually gives way to paralysis and dilatation of the bowel. Visible peristalsis disappears and the dilated coils of small bowel come to lie in parallel loops which can sometimes be seen through thin abdominal wall, as first described by Nothnagel, but can usually be easily identified under the fluoroscope.

The patient has had no bowel movement for two days. No stool comes away on the examining finger and none can be felt per rectum. Twenty vomiting attacks in the first thirty-six hours of an acute appendicitis is an extremely unusual observation. Furthermore, a tendency of the vomiting attacks to increase in number and severity as time goes on, speaks markedly against an appendicitis and in favor of an intestinal obstruction. Of course, a patient who vomits as many as twenty times in two days retains nothing on her stomach and therefore would not be likely to have much if any stool. To be sure the patient has no feculent vomiting yet, but one should not wait for feculent vomiting before making a diagnosis of ileus, if the patient's life is to be saved.

What might an intestinal obstruction be due to at this early age? The patient has never been operated on, nor has she had any serious previous illness. Under such conditions an intussusception, usually ileocaecal, would be the first thing to be thought of. Against this diagnosis, however, is the fact that although these symptoms have

persisted now for thirty-six hours, we still cannot feel a tumor anywhere in the abdomen. There has also been no blood passed per rectum, and none is seen on the examining finger. The severity of the paroxysms of pain is much more marked here than in the average case of acute intussusception. After acute intussusception, the most frequent source of intestinal obstruction in children is a *Meckel's diverticulum* and the various complications arising from it. These forms of obstruction are chronic rather than acute, though occasionally as obstruction becomes complete, the onset may have almost the suddenness of an acute ileus as here.

At any rate the diagnosis of acute surgical abdomen can be made here readily. The next practical point to decide is where to make the incision. Since the principle complaints are located in the right lower quadrant, the choice lies between a right rectus or a right paramedian incision. I shall not decide this point until the patient is anesthetized.

OPERATION

Now that the patient is asleep and relaxed, I am palpating the abdomen to see whether I can feel a mass like an intussusception or a peri-appendiceal inflammatory tumor. I cannot. I believe that I can do all the exploration needed here through a right rectus incision, whereas if the pathology lies far laterally, I may have some little difficulty in reaching it from a right para-median incision (rectus incision).

Dilated ileum presents in the incision. In drawing this out I find here a long band under which about 12 inches of bowel has passed and become constricted, congested, and red, but not hemorrhagic or black.

This band is attached at one end, near the root of the mesentery, and at the other end, where it is thick and fleshy, to the lower ileum. This band is evidently a *Meckel's diverticulum* from its location, size and appearance.

I am cutting the blind mesenteric end between two ligatures. This releases the obstruction completely. This is all that is necessary here, I believe. I now return the loop of bowel. The usual abdominal closure in layers is carried out with catgut and silkworm gut and dressings applied as usual.

POSTOPERATIVE

The next two or three days the patient had a rather stormy time of it with tympany, some vomiting, rapid pulse, and low fever, 99°–100°. Her condition after operation appeared the most serious of the three patients operated. She gradually improved with fluids per rectum and under the breasts, stimulants, heat of the abdomen, etc. The wound healed by primary intention, and the patient left the hospital July 21, apparently fully recovered.

NOTE

The lesson which I wish to draw from these three cases is that while each patient had about the same symptoms, these symptoms occurred in different order and intensity in the three cases. The development and sequence of symptoms are the important diagnostic features in the acute surgical abdomen. *There is no single symptom pathognomonic of acute surgical abdomen.*

Each case requires a careful individual study of the order of development of symptoms and signs, their intensity and interrelations. A history of the patient, hour by hour, from the time of the onset of symptoms, usually brings out the important diagnostic features to best advantage.

First decide whether a given case is surgical or non-surgical. A correct decision on this point is nearly always possible in a well-studied case. If surgical, the next point to decide is where to incise, and this is a less important point. The chief advantage of an attempt at still more accurate diagnosis, and this attempt ought always to be made, is that it tends to make one more observant and consequently more skilled in arriving at the important decision: whether to operate, and where.

OBSERVATIONS REGARDING CERTAIN PHASES OF BRAIN INJURIES

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DURING the past five years a definite advance has been made in the diagnosis and treatment of brain injuries; especially is this true in the field of cranial injuries associated with a lesion of the underlying cerebral tissues and resulting from a direct blow or penetrating missile and of such frequent occurrence in the war. Brain injuries of this type and character are rarely observed in the civil industrial life of peace times, and it is these latter intracranial conditions resulting usually from an indirect cranial injury with and without a fracture of the skull, that will be considered in this paper.

Acute Brain Injuries in Adults.—Progress in the diagnosis and therefore in the treatment of brain injuries has been very much retarded in the past by the mistaken conception of the importance of the presence or not of an associated fracture of the skull; so frequently these patients are designated in the literature and in the hospital records as “a fracture of the base of the skull,” “a possible fracture of the skull,” etc., and yet the presence or not in these patients of an increased intracranial pressure due usually to a cerebral œdema or to an intracranial hemorrhage is rarely considered unless of such degree and severity that the circulatory-regulating mechanism of the medulla is disturbed with the consequent clinical appearance of the signs of medullary compression and, if the intracranial pressure is not lowered, then the signs of medullary œdema itself and—the death of the patient. The presence or not of a fracture of the skull in the patients having an intracranial lesion is of importance in the diagnosis and in the treatment *only* when there is present a depressed fracture of the vault—and these depressed fractures should always be elevated or removed for fear of future complications, and particularly convulsive seizures. The presence or not of a fracture of the base of the skull is rarely of more importance than results from

the increased danger of an infective process extending to the meninges through the middle ear, nares or pharynx—an uncommon intracranial complication, but always to be feared; this is also true of linear fractures of the vault underlying lacerations and severe contusions of the scalp, and also hæmatomata, which may permit the extension intracranially of an infective process to produce a purulent meningitis of varying degree and its complications of abscess formation, etc. Naturally, if careful neurologic examinations do not reveal an increase of the intracranial pressure, as disclosed in the fundi by the ophthalmoscope or in the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer with and without the presence of blood in the cerebrospinal fluid, and no other intracranial lesion can be demonstrated clinically, then the diagnosis of the cranial injury as being one of fracture of the skull (if present) is a proper one in that it is the chief cranial lesion to be ascertained and comparatively of no great danger (Fig. 1, a and b), it is the intracranial lesion, if present, and especially a marked increase of the intracranial pressure—whether due to hemorrhage or cerebral oedema alone—that must be considered in the treatment, both as to the immediate recovery of life and, of almost equal importance in these cases, the future normality of the patient; merely because a patient is able to leave the hospital within three or four weeks following a so-called fracture of the skull does not indicate that this patient is “cured.” Röntgenograms, however, should always be made as a routine procedure upon all patients having cranial injuries of even apparently trivial character; “latent” fractures may thus be disclosed and therefore the expectant palliative treatment of the patient be prolonged rather than the condition be considered of such little severity that the patient is not confined to bed longer than two or three days. Naturally, repeated ophthalmoscopic examinations, and at least one lumbar puncture, should always be performed in order that the true intracranial condition be ascertained as early as possible and the appropriate treatment instituted.

In a series of acute brain injuries in over 600 adults during the past eight years, there was a marked increase of the intracranial pressure in only 30 per cent. of them, and naturally these were the only ones upon whom a cranial operation was performed—that is, only about one-third of the patients having acute brain injuries require

the operation of subtemporal decompression and drainage, whereas the remaining two-thirds of these patients made excellent recoveries with the expectant palliative treatment of absolute rest and quiet, ice-helmet, catharsis and the routine treatment of shock if present. The total mortality in this series of patients was 28 per cent., and if the patients who were admitted to the hospital in a moribund condition of extreme shock, medullary oedema and severe internal injuries are excluded, then the mortality is lowered to only 19 per cent.

The most accurate method of estimating an increase of the intracranial pressure is by means of the spinal mercurial manometer; at the same time the presence or not of blood in the cerebrospinal fluid can be ascertained, the normal pressure is 6-8 mm. of mercury. The expectant palliative treatment usually suffices for these patients whose intracranial pressure does not exceed 16 mm., whereas a pressure exceeding 20 mm. is more safely lowered by an early subtemporal decompression—not after the advanced signs of medullary compression have occurred but in the early stages, whether the clinical signs of medullary compression of slow pulse- and respiration-rates with an increasing blood-pressure have appeared or not; frequently the onset and development of these latter signs are so postponed and then are so rapid that the patient has advanced into the stage of medullary oedema before sufficient time has elapsed to permit a mechanical relief of the increasing intracranial pressure. At best, the pulse- and respiration-rates and an increased blood-pressure are crude and rather late signs of even a marked increase of the intracranial pressure, and of little value in anticipating an acute medullary compression. The ophthalmoscope is usually a much more reliable means of estimating an increase of the intracranial pressure; it is rare, however, to observe the condition of “choked disks” in these patients, as a papilloedema or a swelling of that severity only occurs when the increasing intracranial pressure is of slow character as results from tumor formation or occasionally from a large extradural hemorrhage of gradual formation, otherwise an acute medullary oedema would have been precipitated by the rapid rise of the intracranial pressure and—the early death of the patient before the appearance of “choked disks” would be possible. An oedematous blurring of the nasal halves and of the temporal margins of the optic disks is the usual ophthalmoscopic report in the presence of an increased

intracranial pressure of a height sufficient to indicate a subtemporal decompression as being the safer method of treatment rather than the continuance of the expectant palliative method—not only for the immediate recovery of life, but for the future normality of the patient.

The localization of the intracranial lesion in the treatment of these patients is of little importance compared with the presence or not of a marked increase of the intracranial pressure, whether due to hemorrhage or cerebral oedema. Naturally, in depressed fractures of the vault compressing the underlying cortical cells, large extradural hemorrhage or even the rare condition of small circumscribed supracortical and cortical hemorrhage, these comparatively infrequent lesions should be removed and drained, but in the much larger percentage of intracranial injuries, the hemorrhage (if present) is a diffuse supracortical one and the almost always associated oedema is a diffuse general one, and it is these two factors which cause the increase of the intracranial pressure so that the treatment of these patients should be directed toward a lowering of this increased intracranial pressure, the expectant palliative method being sufficient for over two-thirds of the patients, whereas the operation of subtemporal decompression is only indicated when this increase of intracranial pressure is of marked degree—the remaining one-third of these patients (Figs. 2, 3 and 4). To attempt the removal of small subdural blood-clots through a trephine opening of the vault, and especially in the presence of a high intracranial pressure, is not only meddlesome surgery and of little or no value, but of great danger of permanent damage to the underlying cerebral cortex; whereas, if the increased intracranial pressure is lowered by means of a subtemporal decompression and drainage and, if necessary, by a bilateral decompression (in less than 5 per cent. of these cases), then the natural means of absorption make it possible for the patient to obtain the greatest chance of recovery; besides, through the decompression area itself is afforded an excellent means of exploration and drainage of blood-clots of almost the entire ipsilateral cerebral hemisphere. Even if a cerebral lesion in these patients can be localized—and it very frequently cannot be—it is of little value to expose it, since a large percentage of these lesions are lacerations of the cerebral tissue itself and the lowering of the increased intracranial pressure is the only means of benefiting the patient and obtaining the greatest ultimate improvement.

FIG. 1a.



FIG. 1b.



Extensive multiple fracture of the skull of two patients not associated with a marked increase of the intracranial pressure; therefore, no cranial operation was advisable. Excellent recoveries obtained by the expectant palliative treatment.

FIG. 2.



The vertical incision of the scalp of a right subtemporal decompression—the ideal operation for lowering an increased intracranial pressure and for the drainage of intracranial hemorrhage and excess cerebrospinal fluid. The incision extends from the zygomatic process upward to, but not beyond the parietal crest. The site of attachment for the temporal muscle and fascia, and thus assuring a firm closure.

FIG. 3.



The use of the rongeurs to enlarge the bony opening beneath the temporal muscle to a diameter of at least three inches. The underlying dura is observed to be tense, bulging and pulsating only slightly, due to the increased intradural pressure of hemorrhage and cerebral oedema.

FIG. 4.



Enlarging the dural opening widely and in a stellate manner with the small dural scissors; the spoon-spatula is used to protect the underlying cerebral cortex from possible injury. The dura is not resutured but is allowed to remain open and therefore a permanent decompression is afforded.

The most important and the difficult question in the treatment of brain injuries with or without a fracture of the skull is: "If an operation is advisable, when should it be performed?" This question can more easily be answered by stating the two periods when the operation should *not* be performed. Naturally, we must exclude the majority (about two-thirds) of the patients having brain injuries with and without a fracture of the skull who do not have a definite increase of the intracranial pressure and therefore no cranial operation is indicated. (The depressed fractures of the vault naturally should always be elevated or removed.) The two periods in which an operation is distinctly contra-indicated in cases of brain injury, are, first, the condition of severe shock in the very beginning, and secondly, the condition of medullary collapse—the death knell of the patient. To advise a cranial operation upon a patient—no matter how badly the skull is fractured, nor how extensive the intracranial hemorrhage seems, and that the patient is in the condition of severe shock with a pulse-rate of 120 and higher, then the operation at that period of shock takes away whatever chance the patient may have of surviving the shock: the operation is but an added shock and merely hastens the exitus. No patient having a brain injury should be operated upon in this condition of shock; the mortality is most high, and if a patient does recover from an operation in this period of extreme shock, then he recovers *in spite of* the operation. Cranial operations for brain injuries in this stage of shock were frequently performed in the past and most disastrously, and thus operations were almost discredited in the treatment of brain injuries. The natural reaction following these early operations in the period of severe shock was to wait until there could be no possible doubt that the patient was going to die, unless, as was thought, a cranial operation was performed; that is, the patient was permitted to reach the period of extreme medullary compression—a pulse-rate of 50 and below, irregular Cheyne-Stokes respiration and pulse and profound unconsciousness, before a cranial operation might be considered. This is a most dangerous stage for these patients to reach, and it is doubtful whether recovery can occur even with an operation at this period, the mortality being very high. But if the patient has struggled through this period of medullary compression, and finally reaches the stage of medullary cedema, when the pulse-rate begins to ascend

quickly to 120 and higher, respirations become rapid and shallow—that is, the stage of medullary collapse, then we have the second period when no patient should be operated upon—they all die—operation or no operation. Therefore, if these two extremes can be avoided and the latter of these, medullary collapse, can certainly be anticipated in the operative treatment of brain injuries and their signs cannot be overlooked, then the rational treatment from an operative standpoint depends upon the presence or not of a definite increase of the intracranial pressure, whether there is a fracture of the skull or not; in some of the most serious cases no fracture was present—either to be ascertained at operation in the operated cases, or at autopsy.

Chronic Brain Injuries in Adults.—If depressed fractures of the vault are excluded, then chronic brain injuries are in no way dependent upon the question as to whether the skull had been fractured or not at the time of the cranial injury; just as in acute brain injuries, the presence or not of a linear fracture of the skull is of little importance in estimating the true intracranial condition, the appropriate treatment and the prognosis, so in chronic brain injuries it is of no great value to ascertain that a fracture of the skull had occurred at the time of the original injury, except as an indication of a cranial injury of sufficient force to cause a fracture of the skull; as is well known, however, in many patients following a cranial injury, the skull may not be fractured, and yet the intracranial and cerebral lesion is frequently most severe and dangerous, both to the immediate life of the patient and the future normality. Naturally cranial roentgenograms in court as evidence of a permanent brain injury in these patients is more the result of enthusiastic ignorance than a real conception of the comparative unimportance of the linear fractures themselves.

In order to obtain more accurate data regarding the frequency of chronic brain injuries, I examined, in 1912, the records in three large hospitals in New York City, of their patients having had acute brain injuries during the decade of 1900–1910. The average mortality from the acute brain injury was 50 per cent.; of the patients who survived, following operation or no operation and were discharged as “well” or “cured,” I could only locate 34 per cent. of them in 1912 on account of death from intercurrent disease, change of resi-

dence, and thus "lost," etc.; of these 34 per cent. of recoveries, however, I found that 67 per cent. of them were not well since the head injury, "never the same man again," "always complaining," "cannot do a day's work," "queer ever since," "a bum," "a loafer," and the like; such were some of the minor complaints of both the former patient and the relatives, the latter observing the changes of personality following the cranial injury in a large number of the patients. The complaints of "severe pain in head," "dizzy spells," and very frequently, but still an occasional, patient "having convulsions"—it was this impressive array of symptoms in two-thirds of the patients found, and in many of whom a careful neurological and ophthalmoscopic examination disclosed the definite signs of a persisting intracranial lesion. (At the time of these examinations in 1912, the spinal mercurial manometer was not in use and the importance of an accurate registration of the pressure of the cerebrospinal fluid in patients of this character was not fully appreciated by the medical profession.) Among these post-traumatic and chronic cases there were a number of post-traumatic neuroses, either of the simple associated with business, financial and domestic worries or the complicated type superimposed upon a definite organic intracranial lesion and usually a chronic cerebral oedema of mild degree; other patients exhibited increased and irregular reflexes, an occasional Babinski reflex, impairments of the special senses and the signs of an increased intracranial pressure as disclosed by the ophthalmoscopic examination of the fundi—usually an oedematous blurring of the nasal margins and even the temporal margins of the optic disks—and in the absence of cardio-renal and cardio-vascular disease; the factor of chronic alcoholism so common in many of these patients was excluded as much as possible.

The usual intracranial lesion was apparently a chronic "wet" oedematous condition of the brain following the cranial injury and due to either the result of a supracortical film of hemorrhage, which had not been entirely absorbed, and thus naturally blocking in greater or less degree the normal excretion of the cerebrospinal fluid into the cortical veins and sinuses, or to the continued presence of the acute cerebral oedema immediately following the cranial injury, but in milder degree, owing to its lessened and not complete absorption due to complications in the expectant method of treatment, such as alco-

holism, intestinal and renal toxicemias, emotional strain and other harmful factors in the complete recovery of the patient. The presence of supracortical adhesions resulting from the former subdural hemorrhage were also a factor, and especially in the presence of an increased intracranial pressure with which they were usually associated. These findings were associated in a number of the patients at operation, even at this late date following the acute intracranial injury, and the results have been very beneficial in many of them. Naturally, cerebral lacerations and intracranial lesions destructive of brain tissue cannot be remedied, and the patients are never operated upon unless associated with a definite increase of the intracranial pressure, which should be relieved, and thus a chance for improvement is even possible in these patients by lessening the pressure upon the normal brain cells adjacent to the one primarily destroyed; not only can the signs of impairment be improved, but the symptoms of headache, dizziness, etc., can be relieved, and even entirely removed.

In brief, if depressed fractures of the vault, which should always be elevated or removed, are excluded, only those patients having chronic brain injuries associated with an increased intracranial pressure should be given the benefit of a subtemporal decompression in the hope and belief that a lessening of the increased intracranial pressure will permit a definite and permanent improvement, whereas, those patients in whom there is no increase of intracranial pressure are naturally not operated upon—no matter how extensive the mental or physical impairment is—since the damage in these patients was a primary one occurring at or due to the original brain injury, and the operation of cranial decompression, if indicated at any time, was then rather than months or years later, and especially now in the absence of an increased intracranial pressure. This view cannot be too strongly emphasized because operations are being advised in these later patients with brain injuries in the absence of an increased intracranial pressure, and the results are bad, and they cannot but be bad, since the intracranial pathology cannot now be remedied.

Traumatic Epilepsy.—The condition of post-traumatic epilepsy is a most discouraging one from an operative standpoint, in that it is usually the result of a condition which could have been relieved at the time of the primary cranial injury and thus the epileptiform convulsions could have been avoided. Naturally depressed fractures of

the vault should then be elevated or removed, for if permitted to remain until epilepsy of either the localized Jacksonian type or of the general convulsive type occurs (and it will occur in a large percentage of these patients), then it is frequently too late to obtain a good result, even if the depressed area of bone or foreign body irritating the cerebral cortex is removed, and especially after the so-called epileptic habit (resulting from chronic cortical irritation), has been established; a cranial operation at this late date will in many cases be followed by merely a temporary cessation of the "spells," and within a period of one to three years the convulsive seizures are as numerous if not more frequent than before the operation. In my opinion, it is only those patients in whom the epileptiform attacks are few and of infrequent pressure—not seconding and due to the convulsions themselves (and this can be ascertained by saturating the patient with triple bromides, etc., so that a convulsive seizure does not occur for a period of six weeks, and then estimating and comparing the intracranial pressure accurately by means of the ophthalmoscope and spinal mercurial manometer with the intracranial pressure as registered before this non-convulsive period)—but an increased intracranial pressure which is primary to the convulsions, that is, by an operative removal of the original irritative object, as in depressed fractures of the vault and then a lowering of the increased intracranial pressure and thus a lessening of the cortical cerebral irritation, the patient is given in these selected patients a definite chance of a permanent improvement—if not, in rare cases a cure itself. This careful selection of patients, both as to their general condition of mental and emotional deterioration, the infrequency of the convulsive seizures and the presence of a marked increase of intracranial pressure and not secondary to the convulsions themselves (whether there is a depressed fracture of the skull or not)—these are the comparatively few patients, and the only ones, who can be benefited by the cranial operation of elevation or removal of the depressed area of bone or other foreign body and combined with the operation of cranial decompression. This is late treatment of these patients—the condition should have been avoided and prevented (and it usually can be); many of them become derelicts, so that any treatment, operative or not, cannot make it possible for them to regain their former good health and normality, but in the selected patients as outlined above it is not

only justifiable but the only method now known of affording these patients a chance of recovery.

Brain Injuries in New-born Babies and Children.—In new-born babies, acute brain injuries are usually the result of trauma at the time of parturition, which may be either a difficult one with and without the use of instruments or even a so-called precipitate birth, in that the delivery is an unusually rapid one associated with a rupture of the thin-walled cortical veins; damage to the delicate intracranial structure may also occur in an apparently normal labor; these observations and diagnoses have been frequently confirmed by autopsies.

It has long been recognized that prolonged difficult labor, and especially if instruments for delivery are necessary, is of risk to the immediate recovery of the life of the child; this danger to life itself has been comparatively slight, and if the death of the child did occur, then it was realized (and occasionally confirmed at autopsy) that the intracranial contents had been so badly damaged that even if the baby had recovered, yet it could not have been a normal child—mentally and physically—and therefore it was merely considered an unavoidable and unfortunate result of a difficult labor, the object being to secure a living mother damaged as little as possible and then a living child, if possible. If the child was successfully resuscitated immediately after birth so that it was considered normal and not damaged intracranially, the prognosis as to future normality was naturally excellent—and in fact, this is the normal result. Even if the child was drowsy and stuporous for a period of ten days and longer, when it did not cry as new-born babies ordinarily do, or if it was of the excitable, restless type—crying almost continuously, and whether slight convulsive twitchings of any part of the body were present or not—the condition during an indefinite period of days following delivery was usually a temporary one only so that it was not considered as being permanently harmful to the future of the child; in other words, the child “would grow out of it.” And in the majority of babies with this immediate post-traumatic history the condition does gradually disappear, and fortunately no ill-effects are later to be observed due to the entire absorption of the intracranial hemorrhage and cerebral oedema. There is a small percentage of babies, however, in which this happy result does not occur; either they remain in a comatose condition with and without convulsive

seizures for several days and then die (and at autopsy an extensive subdural and usually a supracortical layer of hemorrhage is revealed, together with a very "wet" oedematous condition of the brain) or they apparently became normal and were considered so until the sixth, seventh, eighth or ninth month later, when it is realized that the child is not developing as a normal child should—is not holding up its head, does not attempt to grasp and to hold things, notices little if anything, etc., and as it becomes older this retardation and impairment, both physically and mentally, becomes more marked; even at this late date of months, and especially without careful examinations, the parents may be told that nothing is really "wrong" with the child, "merely retarded," and "it will grow out of it." These children rarely do, however, when the condition is the result of a large intracranial lesion at the time of birth—usually a supracortical layer of hemorrhage with little or no primary damage to the brain itself, and in the babies which cannot "take care of" this large amount of hemorrhage and cerebral oedema by the natural means of absorption, then the effects of this intracranial condition associated with a definite increase of the intracranial pressure are later shown in a general retardation of the development of the child, both mentally and physically. Unless this increased intracranial pressure is relieved early—if not immediately after birth, then within several days, or if the condition is permitted to continue until the latter months of the first year and even later, then the lowering by means of a subtemporal decompression, and if necessary, then a bilateral decompression—although the longer this increased intracranial pressure is allowed to continue, either through ignorance, carelessness or mistaken diagnosis and judgment, just as much more permanent is the cerebral impairment in its mental and physical results. The differential diagnosis at this late date is between that of lack of development of the cerebral cortex or its pyramidal tract fibres (of the so-called Little's disease), or meningitic and meningo-encephalitic distinctive forces associated or not with embolic or thrombotic complications, hereditary lues (less than 2 per cent.), and then the condition of intracranial hemorrhage at the time of birth and of much larger amount that the natural means of absorption have not sufficed to permit the normal lowering of the intracranial pressure of hemorrhage and chronic cerebral oedema resulting from a partial blockage of the stomata of exit of the cerebro-

spinal fluid in the cortical veins, sinuses, etc., by the origination of this layer of supracortical hemorrhage; and thus, in reality producing a mild external hydrocephalus similar to the condition of hydrocephalus—but in milder form resulting from an extensive meningitis process which does not block the ventricles, and therefore producing the more common type of external hydrocephalus. These chronic brain injuries occurring in children who become impaired both mentally and physically, and particularly of the type of cerebral spastic paralysis, are of greater frequency than has been generally recognized.

In the acute brain injuries, with and without a fracture of the skull occurring in children under twelve years of age, the immediate effects of intracranial lesions can be withstood much more successfully than in adults—the initial shock is less, the reaction is much stronger and prolonged and they can recover from intracranial trauma as far as immediate life is concerned much more easily and with fewer immediate complications than is possible in adults; but the remote effects, however, of serious and prolonged intracranial lesions in children are more permanent in these patients later in life and they form a very influential factor in the future development of the child—both mentally and physically. It is this remote factor and result of intracranial injuries in childhood which have been rather neglected and overlooked.

Acute Brain Injuries in New-born Babies.—It is not uncommon for acute brain injuries to occur in new-born babies during parturition—usually a difficult labor with and without the use of instruments, but it is rare for these intracranial lesions to occur associated with a fracture of the skull; occasionally depressed fractures of the vault and of the so-called “ping-pong” type result, but it is not unusual for a frank linear fracture of the flexible newly-formed bone to be demonstrated, either by röntgenograms, operation or at autopsy. If there is present in these new-born babies any abnormality of the base of the skull, then it is almost invariably a diastasis and separation of the suture lines with and without their overlapping, one over the other; the suture line most frequently involved is that median one between the two parietal bones and overlying the longitudinal sinus, which may thus be torn, permitting an intracranial hemorrhage of varying size to form over the cortex of one or both hemispheres of the brain—and this is the most common type of intra-

cranial hemorrhage occurring in new-born babies as a result of the change of continuity of the bones of the vault. This separation of the suture lines and the overlapping of the adjacent bones rarely persists after birth longer than hours or days at the most, during the active second and third stages of labor, and then the bones resume their normal relation and position, but after the damage to the sinus has resulted. This is the reason why careful bimanual examination of the head of these children and still later röntgenogram planes only infrequently demonstrate the presence of the overlapping of the lines of suture. The frontal bone in its posterior relation to the parietal bones to form the canal suture, and the occipital bone in its anterior relation to the parietal bones to form the lambdoidal suture, are the next most common sites for the overlapping of their respective suture lines, and yet intracranial lesions only occasionally follow since there are here no underlying sinuses.

If the longitudinal sinus is not torn, and it is possibly one of the most frequent causes of the condition, then the next most usual source of the supracortical hemorrhage is a rupture of the delicate supracortical veins of either or both cerebral hemispheres as a result of a severe venous stasis and congestion occurring during a prolonged difficult labor; the hemorrhage may be only a local one—the size of a ten-cent piece or a silver quarter, and yet the associated cerebral oedema following the cerebral trauma is always present, and may in many patients be the more serious factor. It is thus seen that the intracranial hemorrhage in these new-born babies rarely occurs in the cerebral cortex and in the brain itself and therefore causing a primary destruction of brain tissue (and no regeneration), but the hemorrhage is almost always subdural but supracortical—lying upon the surface of the brain—and its damage to the underlying cerebral cortex is one of pressure, due both to the hemorrhage itself and to the resulting cerebral oedema; that is, if this supracortical hemorrhage and excess cerebrospinal fluid can be successfully drained and thereby the increased intracranial pressure be permanently lowered, then these babies will have an excellent opportunity to recover not only the immediate recovery of life, but that of future normality, and now is the ideal time for the appropriate treatment of these patients; later in life the impaired condition can be improved, but rarely is a perfectly normal child then possible.

No doubt there are many cases of latent intracranial hemorrhage at birth where there are no marked clinical signs of the presence of the lesion and where the natural means of absorption are sufficient to "take care of" the mild increase of the intracranial pressure—and a normal child is possible. And on the contrary, later impairments occurring in certain children in adolescence, such as mild mental retardation, emotional instability, and even epilepsy itself, may be due to a mild clinical sign not recognized or overlooked, and the later appearance of signs indicative of a former intracranial lesion with resulting adhesions, etc., and it is then usually too late to obtain a satisfactory result by any treatment now known. The treatment should be preventative whenever possible.

Although the labor itself, in these babies developing as intracranial hemorrhage at the time of birth, need not be a prolonged difficult one associated with the use of forceps, yet it very frequently is; also, the condition itself occurs most often in first babies of full term. The condition occasionally results even from a so-called normal delivery, although a difficult labor with and without the use of instruments is the usual history obtained. Any new-born baby which does not behave normally within the first two or three days after birth in that it is unusually drowsy and even stuporous, and especially in the presence of convulsive twitchings of any part of the body, that baby should be carefully examined for definite signs of an intracranial hemorrhage; a lumbar puncture is of the greatest diagnostic importance. If free blood is found in the cerebrospinal fluid, not only is the diagnosis confirmed, but an excellent means of drainage is thus afforded unless the pressure is high—over 15 mm., as registered by the spinal mercurial manometer and associated with tense fontanelles and positive ophthalmoscopic findings. Daily repeated lumbar punctures with removal of 10–12 c.c. of bloody cerebrospinal fluid may be performed upon a number of consecutive days, until the pressure of the cerebrospinal fluid does not exceed 10 mm., and in these patients an excellent result is frequently obtained. In those babies, however, in whom the increased pressure of the cerebrospinal fluid reaches a height of 15 mm. and even higher, and especially when associated with tense fontanelles and positive ophthalmoscopic findings of increased intracranial pressure, then a modified subtemporal decompres-

sion and drainage is most advisable in order to obtain not only a living child, but of the greatest importance—a normal child later.

Acute Brain Injuries in Children.—In children under twelve years of age, cranial injuries may be of comparatively trivial character, and yet the most serious intracranial lesions often result, with and without a fracture of the skull. In these patients, however, a fracture of the vault, and even of the base, occurs much more easily than in adults, and the relative importance of the fracture of the skull in brain injuries is not illustrated better than in a study of these patients.

Not only do children withstand better the immediate effects of the cranial injury, and especially the severity of the initial shock in that their reaction is a more vigorous one and thus assuring a higher percentage of immediate recovery of life, but it seems that the cardiac and respiratory centres in the medullar are more resistant and their circulatory mechanism more adaptable to sudden increases of the intracranial pressure; it is for this latter reason that the expectant palliative treatment can be used successfully in a larger percentage of children having brain injuries, both as to the immediate recovery of life and to the future normality, than is possible in adults in whom not only is the initial shock a most serious factor, but the sudden increase of the intracranial pressure is an only too frequent cause for early medullary complications of compression, and even medullary oedema itself. In this series of brain injuries in children under twelve years of age the expectant palliative method of treatment is alone sufficient and eminently satisfactory in over three-fourths of these patients, whereas the operative treatment to lower a high intracranial pressure, whether due to hemorrhage or excess of cerebrospinal fluid, by means of a subtemporal decompression and drainage is only indicated in about one-fourth of the total number of these patients; naturally, and it has been repeated a number of times in this book, all depressed fractures of the vault should be elevated (and this is more frequently possible than in adults) or removed, for fear of future complications, and chiefly that of cortical irritation with its resulting emotional instability, and even epileptiform seizures.

Cranial injuries apparently of very trivial character and of such slight importance at the time of the "bump" may cause an intracranial lesion of the greatest danger—not only to the immediate life

of the child, but also in its remote effects, later in life, upon the normal development—mentally, emotionally and physically.

Chronic Brain Injuries Occurring in Children.—The persistent effects of brain injuries occurring in children between the ages of two and twelve years are very similar to those occurring in adults with the important exception that since the mental and emotional “make-up” of the children is in the process of development, any prolonged impairment of function during this formative period is later exhibited in a greater retarded mental and even physical condition; especially is an emotional instability to be feared, and if a definite cortical irritation is present to a degree that a mild chronic cerebral oedema exists, then the danger of epileptiform seizures is one of not only great frequency but of the direct consequences to the patient: if once convulsions occur, and especially if months and years after the brain injury, then the chances of benefiting the patient are just that much lessened, and the longer the convulsions persist the greater the improbability of any procedure being of assistance to the patient; on the contrary, epileptiform seizures occurring at the times of the acute intracranial condition or within a short period following it—these patients are frequently restored to a normal mental and emotional condition by the appropriate medical treatment.

Notwithstanding the fact that children withstand the acute effects of brain injuries much more easily than do adults, and particularly is this true of severe conditions of initial shock and high intracranial pressure, many children having brain injuries have been carelessly treated and the remote effects of the intracranial lesion have been overlooked, merely because the patient has made an immediate recovery of life; this latter result is all-important, but the future normality and good health of the child should also be considered. It has been recognized for a number of years that all depressed fractures of the vault should be elevated or removed at the time of the acute injury—not for the immediate benefit to the patient, but to lessen the danger of future impairment and complications, and especially emotional instability and epileptiform seizures; this routine method of treatment has been advocated chiefly in children on account of their developmental period of life when any intracranial lesion, however insignificant its present symptoms and signs may be, in its remote effects is frequently of a most serious character. The significance, however, of a persistent

and chronic increase of the intracranial pressure following brain injuries in children, whose apparent excellent recovery from the immediate effects of the injury has been complete, has been overlooked, and it is only by examining these patients over a period of years that it is possible to state that these children—and they form less than 20 per cent. of all patients injured—do not later develop mentally, emotionally and physically as they should on account of the effects of a prolonged mild increase of the intracranial pressure; an emotional instability is possibly the most common result, a mental retardation, and, as stated before, the great danger of epilepsy itself. This chronic cerebral œdema persists in children less frequently than it does in adults, but its ultimate effects are more pronounced in children on account of the immediate development of the cerebral nerve cells in all their activity; it is thus realized that the resulting functional impairment due to an increased intracranial pressure if prolonged over a period of months and years, may produce a definite organic change of tissue—and it is then irreparable. The treatment should be the appropriate one at the time of the acute injury and not months and years later—when frequently only an improvement can be obtained and not a normal individual.

INCOMPLETE UNION AND MAL-UNION OF FRACTURES

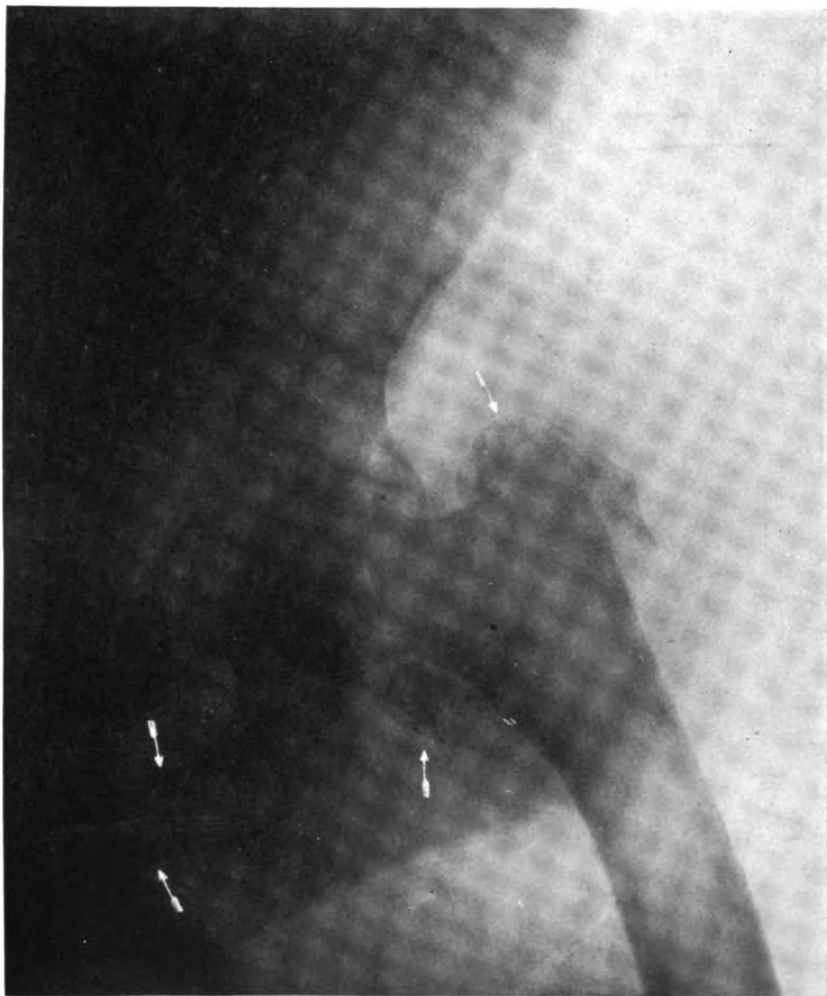
By JOHN B. ROBERTS, M.D.

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IMPERFECT, or incomplete, union of the fragments of a fracture is to be deprecated. It means impairment in a greater or less degree of the patient's bone efficiency. Mal-union, if only a slight alteration in anatomical outline, is not necessarily a disability. If it occur in childhood or adolescence in the shaft of a bone, it may be compensated for or entirely disappear during growth to adult years. Much depends, however, on the character and site of the mal-union, as well as the degree of anatomical inaccuracy of outline. Deformed union of a nasal bone may be a cosmetic defect of importance and functionally of marked importance by reason of interference with respiration. Secondary results may arise in children, causing mouth breathing, imperfect dental occlusion and interference with the shape, features and general health. On the other hand, an abnormal curve in the shaft of the femur, even of considerable degree, in a growing child may straighten out in succeeding years and leave no lameness or marked inequality in femoral symmetry.

Mal-union of a fracture may be imperfect or vicious because consolidation has occurred with abnormal position of the fragments, thereby changing the bone's shape, or because no rigid bond of union has been developed to give normal rigidity at the site of fracture. The first condition of anatomical deformity may be objectionable merely because beauty of contour has been sacrificed, a loss of minor importance in some regions of the skeleton; but a functional disability from change in bony outlines, which restricts motion or other normal usefulness, is of greater moment. A joint may be shocked by irregularity within or near it, a limb shortened, the axes of bones altered, or the mechanical efficiency of the skeleton lessened by changes in the contact planes of articulating surfaces, or by impairment of the functional ability of the surrounding soft parts. The terms, deformed

FIG. 1.

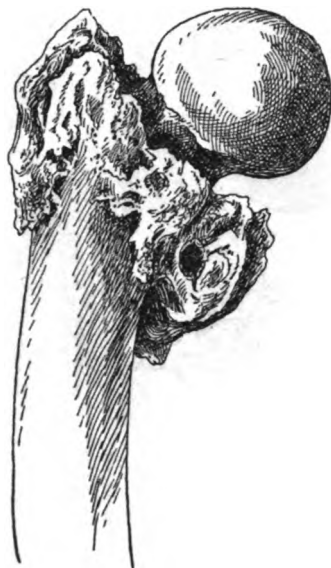


S. B.—Fracture of tubes and ischium; also at base of neck and both trochanters of femur in an insane woman, with cardiac dyspnoea. Impossible to carry out proper surgical treatment. Death about 2 months after injury.

union or mal-union, have been used to describe these vicious deviations from satisfactory healing.

Failure to reduce a fracture at the beginning of treatment may result in mal-union, non-union, or combination of both. This is evident from the recognized requirement of coaptation, if proper alignment, freedom from overriding, and absence of rotary displacement are to be secured. Infrequent examination and consequent failure to notice and remedy deviations from proper contour while callus is soft, is a pregnant cause of mal-union. In this variety of imperfect union,

FIG. 2.



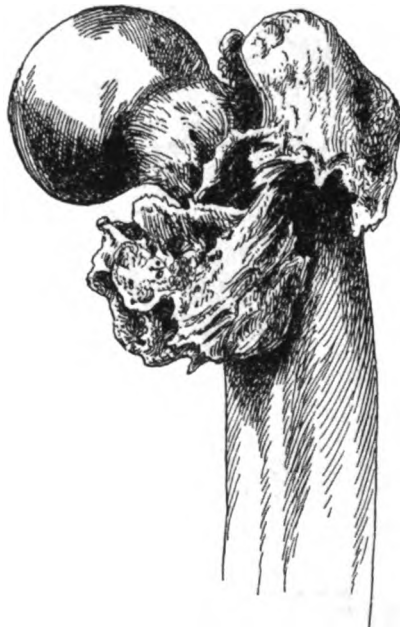
S. B.—Fracture of base of neck of femur and both trochanters in insane woman who died of cardiac disease about two months after injury. Anterior view. Note deformity and exuberant callus.

therefore, early reduction and frequent removal of dressings in the early days of treatment are essentials. Similar activity and wide-awakeness are important qualities of the fracture surgeon, if delayed union, non-union, or development of true pseudarthrosis is to be recognized early enough to avert the undesirable occurrence or to relieve the resulting disability. In a word, deformed union in closed uncomplicated fractures is usually due to lack of knowledge of efficient treatment of the fracture or to neglect in applying well-known mechanical and physical principles to the problem of treatment.

There are, of course, cases, not a few in number, in which intractability of the patient, complicated character of the injury or uncontrollable conditions render perfection in treatment impossible. Deformed union of various degree is more frequently seen than non-union.

A second possible deviation from the usual process in repair of sundered bones is delay in ossification of the fibrocartilaginous tissue, the so-called callus, deposited between and around the fragments of bone. Nature uses this callus to maintain contiguity and relative immobility of the pieces until the fibres of the apposed surfaces have

FIG. 3.



S. B.—Posterior view. Compare with Fig. 2.

become rigidly joined by the osteoblasts. The divergent views of pathologists as to the parts played by periosteum, endosteum, marrow and contents of the Haversian system in union of fractures need not be discussed in this consideration of imperfect unions.

Delay in union may be due to some abnormal condition of the patient's blood and solid tissues. In such cases, union occurs after a longer convalescence than is experienced in similar fractures in other persons. The question of moment is the discovery of the cause,

psychic or physical, mechanical or metabolic, of the delay. A query, however, also arises in the surgeon's mind as to when the delay has been sufficiently protracted to become an urgent reason for adopting the treatment proper for a true non-union.

The most troublesome form of defective consolidation is absolute failure of the bone to reestablish its continuity and give normal skeletal rigidity. The bone ends may atrophy and become pointed and compact; or there may be created a bursal sac between the opposing sur-

FIG. 4.



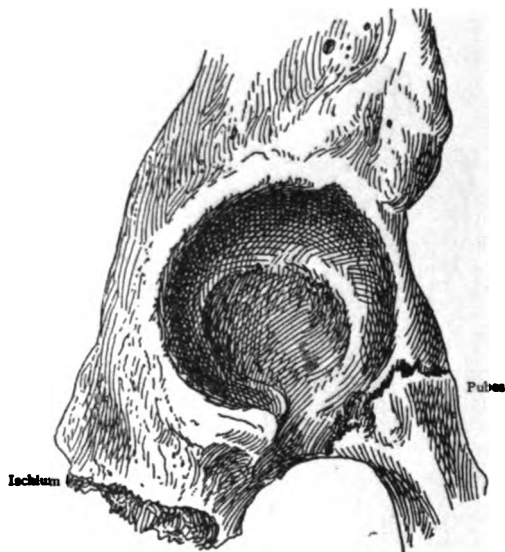
S. B.—View of articular surface. Compare with Figs. 1, 2, 3.

faces, resembling a true synovial joint cavity. The latter is the so-called false joint, which may be differentiated from absolute non-union. This exists when two wedge-shaped, or pointed, ends lie near together with no bond of union whatever or only a flail-like band of fibrous ribbon between them.

It seems likely that the atrophic condition occurs when there is defective metabolism of the patient, or a decided loss of bone structure, from comminution or necrosis, preventing contact even after

reduction. Inability to get the ends of the bone in contact because masses of muscle or tendon lie between the broken surfaces will explain many instances of false-joint (pseudarthrosis). The musculo-fibrous tissue prevents actual contact of bone ends, the osteoblastic cells are unable to cement the fragments together, and the resulting mobility and friction give rise to a bursa to lessen friction. Infected fractures, especially those due to gunshot injury, are very liable to delayed or deformed union.

FIG. 5.



S. B.—Fracture of pubes and fractured end of ischium. Compare with Figs 1, 2, 3, 4.

Association of deformity with the failure of union would be expected when pseudarthrosis has developed as a result of entanglement of fragments in extensively lacerated muscles. When a fragment has been thrust through a buttonhole made in a muscle by its sharp end, atrophic non-union would be rather likely to be found, especially if the surgeon has failed to withdraw the perforating piece of bone from the muscle belly so as to bring it into contact with its fellow fragment.

It will be seen, therefore, that imperfections in the consolidation of fractures may occur as delay in union, deformed union, production of a joint-like union, total lack of union or as a combination of these defects in cure.

FIG. 6.

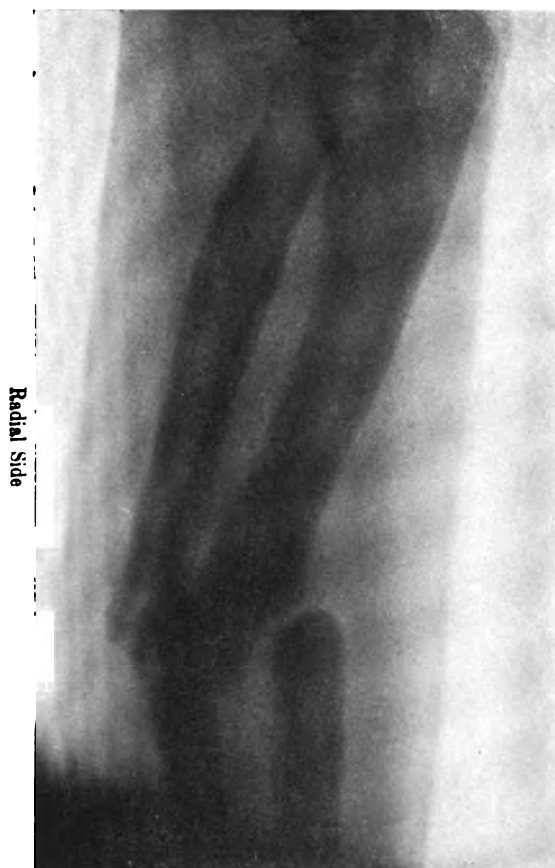
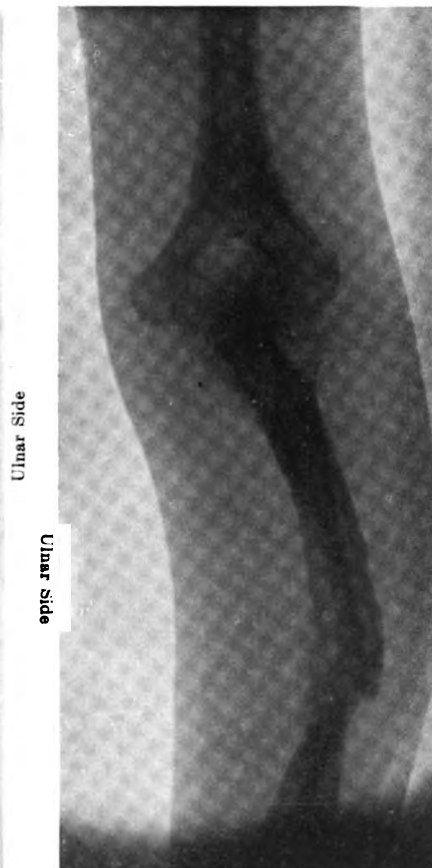


FIG. 7.



Lateral view of ununited and deformed fractures of shafts of radius and ulna with rotation and angulation of fragments. Hand fixed in supination. Operation disclosed bridges of bone between upper fragments of radius and ulna, and only fibrous tissue between the fragments of the two bones.

Ununited and deformed fracture of radius and ulna. Antero-posterior view before operation. Compare Figs. 6 and 7.

FIG. 8.



Ununited and deformed fracture of radius and ulna four months after operation by aluminum plate on ulna, with steel screws, kangaroo tendon suture in radius (intramedullary). Radius wrapped with graft of fascia lata. Shows some union of ulna; no union of radius. Anterior view of elbow joint which was partially ankylosed. Lower fragments in mid-pronation; upper fragments in full supination. Compare Figs. 6 and 7.

FIG. 9.

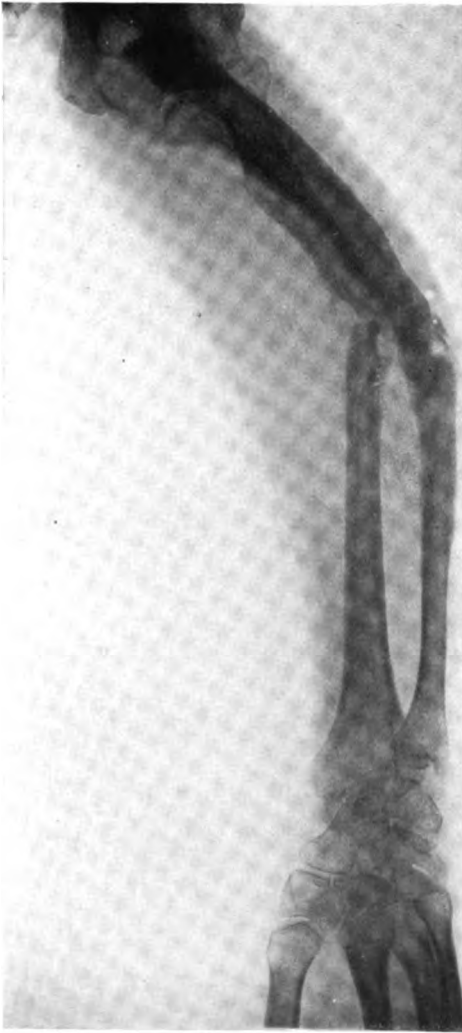
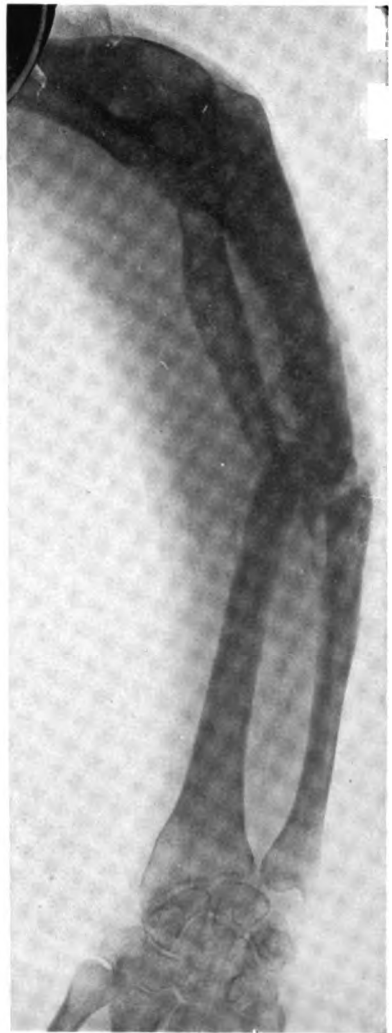


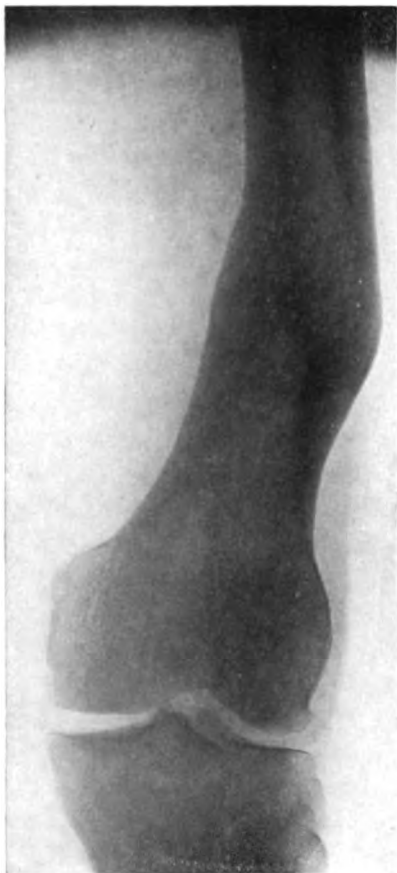
FIG. 10.



Non-union of fracture of radius and ulna after unsuccessful operation with metal plate. Deformity was angular and rotary. Plate was put on ulna and kangaroo tendon suture and fascia lata wrapping used for radius. Compare Fig. 10 (antero-posterior view).

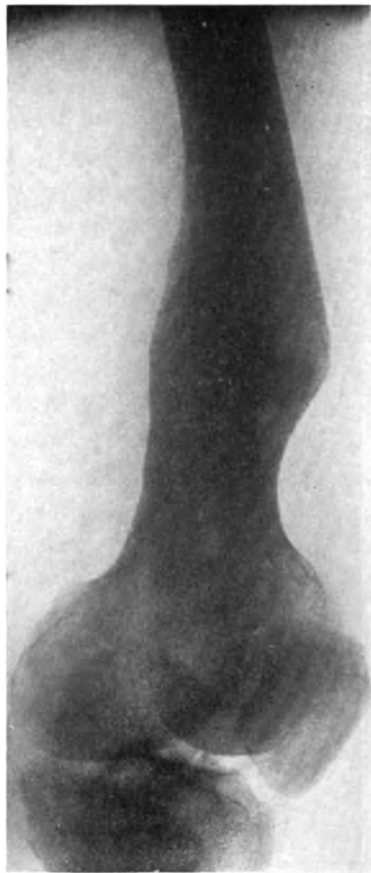
Same case as in Fig. 9. (Lateral view.)

FIG. 11.



Old malunion of femur with displacement inwards (anterior view). Compare Fig. 12.

FIG. 12.



Old malunion of femur showing backward displacement of lower fragment. Lateral view. Compare Fig. 11.

Deformed Union.—Subcutaneous refracture for the correction of angular deformity of the union of shaft fractures is comparatively easy for from three to six months after injury. The cross-breaking strain is readily applied by grasping the ends of the bones and using the viciously united fragments as levers. When necessary, a wedge-shaped fulcrum may be applied beneath the convexity of the union to localize the fracturing force. The operator's bent knee or a padded block is available. In long-standing deformity of big bones like the femur, it may be necessary to increase the operator's manual power by bandaging a flat board to the limb so as to stiffen the distal joint and thus lengthen the lever to which the power is to be applied.

Occasionally simultaneous traction upon the distal portion of the limb may aid. Perhaps this is more valuable when the angular distortion is combined with overlapping. The clove hitch or the Levis traction plate well supplies the attachment to the limb. Radiographic examination of the osseous bond will give a fair idea of where and how the breaking strain should be applied. Driving an osteotome between the fragments in one or two places will weaken the connection and render the refracture possible with less expenditure of force. The small incision in the soft parts, as in correction of knock-knee and bowleg, need not destroy the practically subcutaneous character of the operation.

Osteoclasts are seldom to be used in these days of successful aseptic open operations. Conditions may arise, however, in which these machines are to be preferred. Such must be unusual.

The last-named aids to the manual power of the skilled operator are more likely to be called into play when the deformity is caused by overlapping or rotary displacement of the fragment ends. Such deformities, too, are the ones in which subcutaneous rupture may be unsatisfactory. It is in these instances that open osteotomy is most frequently to be selected.

Subcutaneous correction of vicious union is available and successful for a long period after the fragments have been firmly united. Deformity from unreduced fracture at the lower end of the radius may be corrected thus at four to six months. Firm union of deformed fracture of the shaft of the femur may be ruptured subcutaneously up to six months. The time of successful disruption will depend upon the character of the displacement, the method of applying the power,

and the familiarity of the surgeon with the methods. Recently the bandaging of a board to stiffen the knee gave one of us leverage enough to break up a badly united femur impossible to manage with the hands alone. The after-treatment is to be conducted on the same lines as are employed in similar accidental fractures. The surgeon will often be aided in the choice of method by a careful study of the causes of failure to obtain proper coaptation in the original bone injury.

In delayed union, deposits of callus should be hastened by massage of the limb, good food, fresh air, happy surroundings, hopeful attendants, and congestion of the limb by continuous or interrupted use of rubber constriction on the proximal side of the fracture. Calcium carbonate, grains v-x, and calcium lactophosphate, grains v-x, in powder, three or four times a day, as an internal remedy, or calcium chloride may be useful.

There is the need of due appreciation of the value of psychic as well as mechanical and medicinal agents in the surgery of delayed union. This is the reason that sometimes operative attack is too hastily undertaken for the treatment of delay in consolidation of fractures.

Supplementary helps in securing callus formation in delayed union are massage, hot water bathing, permitting some use of the limb, passive congestion with rubber constriction above the break, rubbing the ends together by manipulation, and injecting blood hypodermically into the tissues around the fracture.

When it is evident that the delayed union is likely to be permanent, treatment for non-union must be adopted. It should be remembered that experience seems to show that direct fixation with metal plates retards consolidation of fractures. Hence, former views of the time limit allowed for consolidation before accepting the diagnosis of non-union should be somewhat lengthened in recent fractures treated with metal plates.

Non-union and actual false joint present great difficulties in successful treatment. They are fortunately rather unusual occurrences in uncomplicated fractures. Patients in poor health when the fracture was received, persons with multiple fractures due to very serious traumatism, are more liable to deficient callus and unattained ossific consolidation of the bone injuries.

Interposition of soft parts between the fragments, infection, insuffi-

cient arterial supply and wide separation of fragments or loss of considerable bone substance by the injury or secondary necrosis are effective in producing pseudarthrosis or entire absence of union in even those previously in vigorous health.

Bad general condition of the patient leading to inefficient metabolic activity at the site of fracture may be responsible; or local malignant disease or osteomalacia may be present. Imperfect coaptation preventing osseous contact, a great degree of muscular displacement and perhaps faulty immobilization are possible factors in the production of a mere fibrous union or of a false joint. Delayed union is more apt to be due to deterioration of vital forces; but non-union and false joints nearly always depend upon a local cause. This is apt to be mechanical. A fracture that remains painful long after union is apparently obtained will usually be found on close examination to be the seat of a slight degree of abnormal mobility. Ununited fracture has in many instances this accompaniment of pain on use of the limb.

When the treatment suitable for delayed union has been tried, or radiographic examination shows that diagnosis of non-union is applicable, operative treatment may be said to be demanded. This aims at removing the local cause and stimulating fundamental activity; or, at converting the old ununited fracture into a recent one with fresh surfaces placed in contact with each other.

The subcutaneous method is to violently bend and rotate the fragments so as to forcibly tear asunder the fibrous connections and strongly rub the ends together. This may be followed by tenderness, swelling and consolidation. The manipulation to be thorough requires anaesthesia, and should be of a character to bend the fragments at a right angle, rotate and extend as much as safety to vessels and nerves will allow. Immobilization with rigid external support is the after-treatment.

Aseptic precautions have made exposure of the seat of non-union so safe that now the surgeon rarely delays long before attacking the ununited fracture through a free incision. This allows more accurate information as to the local cause of the failure of union, and a wiser and more successful direct management of the condition.

In pseudarthrosis open operation is the only efficient treatment and even in the less perfect illustrations of false joint it is often to be adopted. The fragments are freed from fibrous muscular tissues,

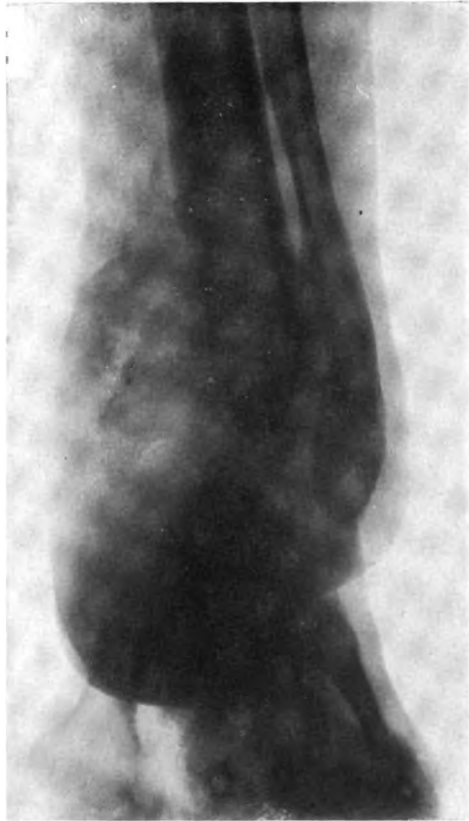
the ends, often atrophied, sawed off, and placed in contact. If atrophied, the fragment must be exsected at a point well above the narrowed compact portion so as to expose a wide surface of cancellous bone. The bare surfaces should be placed in contact, or a graft of bone, preferably autogenous, may be placed between the freshened ends. Coaptation should be obtained and may be maintained by an autogenous inlay graft, as suggested by Albee; or, by pegs or dowels of bone, or plates, screws, wires or catgut mata. It is possible that the suggestion of using fascia from the fascia lata of the thigh as a sheath or cuff to maintain the contact may prove valuable. External support by a gypsum splint or encasement is used as in accidental fractures for the maintenance of correct position of the fragments. This should be continued for many weeks, for hasty mobility is usually an error in operative surgery of ununited fractures. In fracture of one of the parallel bones of the forearm it should be seen that the resected bone is not prevented from contact because the one which has not been broken is longer. In such cases the gap between the fragments should be bridged by an autogenous graft from the patient's tibia, a dowel graft driven into the medullary canal of the two ends, or a wedge-shape graft driven between the fragment ends.

It has been suggested that when repair is likely to be inactive, the wound might be left open and packed with sterile gauze to cause irritative increase of metabolic changes. The surgeon should always see that there is contact of considerable surfaces of cancellous bone; and look to giving the patient happiness, fresh air and good food. Too little attention to these hygienic factors is a common fault among us surgeons. A good judge is said to need his law tempered with justice and mercy. A fracture surgeon needs his mechanics tinctured with psychology, physiology, and hygiene.

Combination of deformed union and non-union is apt to be a most troublesome problem for surgical repair. An elbow may be shocked, the forearm bones replaced by muscle at the seat of fracture and the fragments ununited and atrophied. The femur and patella may be broken, one of the fractures being open, the other closed, and the patient the subject of grave malnutrition and despondent mind. Such problems require patience, enthusiasm, faith, skill and time to bring about the hoped for convalescence.

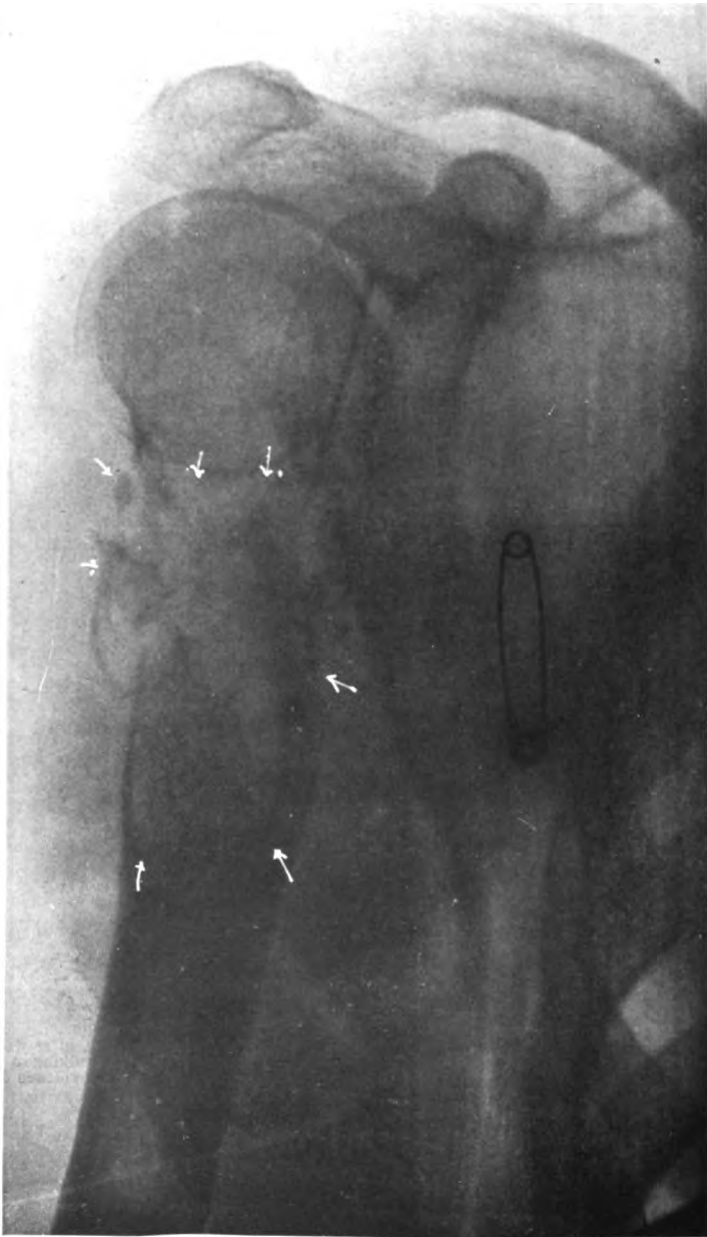
If an operation for non-union has failed to give the patient a

FIG. 13.



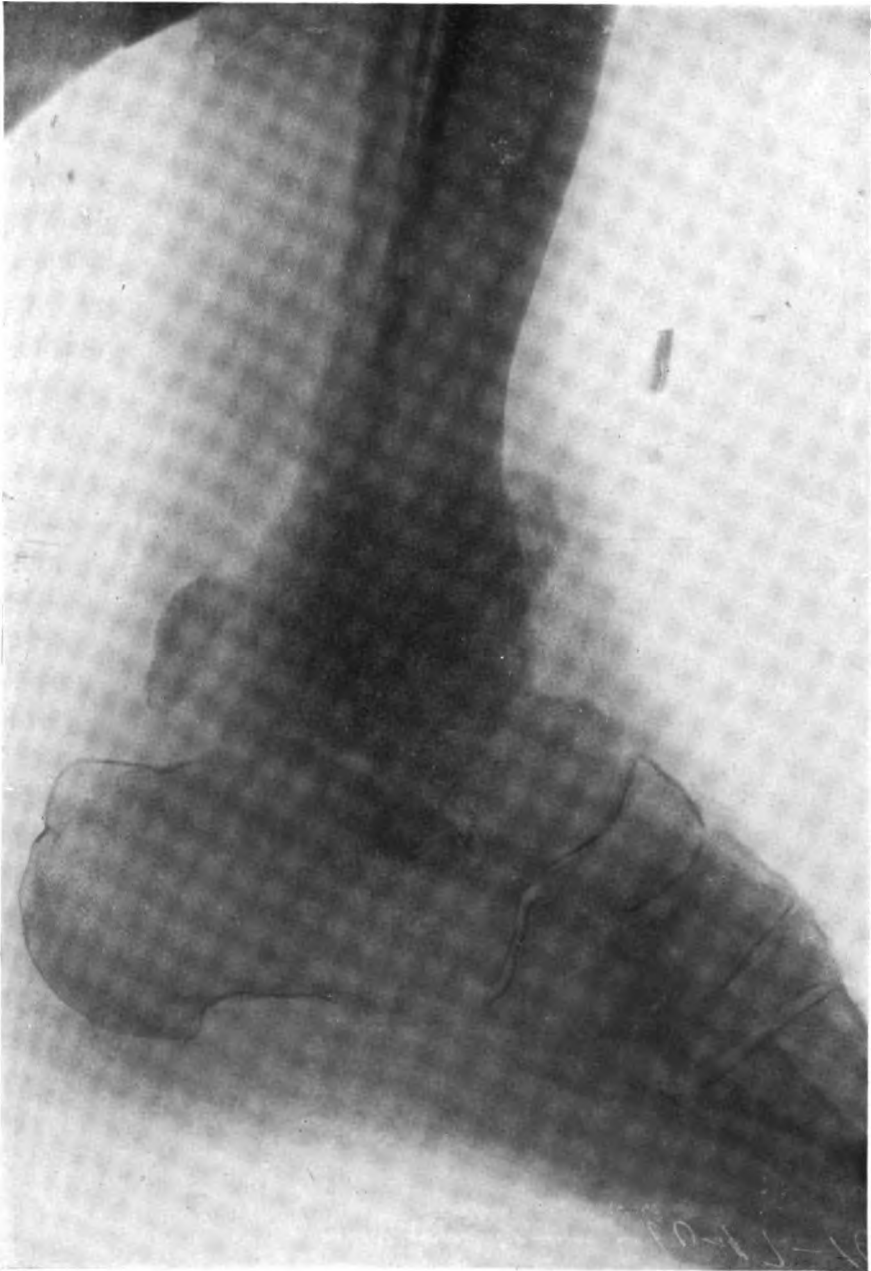
Great deformity following too early weight bearing by discarding crutches, after well united fracture of lower end of tibia and fibula. Fracture occurred spontaneously while walking in a patient under treatment for specific arthropathies of knees. Röntgenograms showed no evidence of special disease of bone at ankle at time of fracture. Anterior view. Right leg.

FIG. 14.



Cystic degeneration of humerus permitting pathological fracture. A man, aged 32 years, sustained fracture by fall against a chain; not a heavy blow. Had had pain in right humerus previously. Fracture was diagnosed by preternatural mobility with soft or almost no crepitus. Wassermann negative. Bone united without difficulty, though patient was given potassium iodide with mercury. Case may prove to be sarcoma.

FIG. 15.



Old fracture (eversion) of lower end of tibia and fibula due probably to syphilis. Malunion due to too early discarding of crutches. Union at the end of about two months was satisfactory without deformity. Antisyphilitic treatment was employed before and after fracture. (Lateral view). Compare antero-posterior view.

firm union, after careful abstinence from meddlesome mobility and the judicious general and psychic treatment advocated above, the surgeon usually should allow a period of several months to elapse before undertaking another open operation. This is especially true in fractures which have been infected originally or after the operation for relief of the non-union.

Infected open fractures are especially prone to deformed union, delayed union and non-union. Such fractures, and fractures in which operative fixation has been followed by infection and failure in repair, need a far longer period of fixation and abstinence from operative disturbance than do those which have been given time for the previous infecting organisms to lose virulence.

In operating upon ununited fractures, where there is a pseudarthrosis, it is usually wise to dissect away very thoroughly the false capsule and the synovial bursa between the fragments. The compact tissue at the ends of the bone constituting the false joint should be reamed out in order to expose the medullary cavity and the osteoblasts, from which new bone is expected to develop. Bone pegs cut from the tibia may then be inserted into the medullary cavity and external fixation used by means of a gypsum encasement. In the X-ray study of the progress towards union after operations for ununited fracture, it may be observed that the transplant, whether a dowel or an inlay, has become united by bony growth at only one end. Should the condition of non-union persist, as shown by repeated X-ray examinations, and union take place at the other end of the graft, it is proper after the lapse of a few months to reopen the wound. An operation should then be performed to obtain contact between the ununited end of the graft and the fragment at the end of which union has not occurred.

In non-union operations, it is sometimes well to use wire or a Lane plate for direct fixation at the time of operation, in addition to the steadying of the grafted region with the external plaster-of-Paris encasement. In difficult cases much stress must be laid upon the necessity for a very prolonged fixation in the gypsum case. This is because many patients fail to get union on account of the fact that they desire to hurry up motion in the neighboring joints. Prolonged fixation of the seat of operation for many months is often essential for obtaining union. Early removal of the cast or attempts at motion at the adjacent joints may lead to absolute failure of the grafting operation.

HYPERTROPHIED ANAL PAPILLÆ (PAPILLITIS)

By CHARLES J. DRUECK, M.D.

Associate Professor of Rectal Diseases, Post Graduate Medical School and Hospital, Rectal Surgeon to Peoples Hospital, Chicago

THE transition from mucous membrane lining the intestinal canal to the external skin at the anus is accomplished in three distinct steps each forming a distinct zone. In the intermediate zone the epithelium changes to several layers of polygonal cells. Dermal papillæ are found here. The submucous layer is very vascular and contains a ganglionated plexus together with lamellar corpuscles. The muscular coat terminates in slender bundles in the rectal columns forming the internal dilator muscle of the anus, while the circular layer of the muscular coat becomes thickened into the internal anal sphincter. This internal anal sphincter is of smooth muscle fibres. Below this muscle is the external anal sphincter which is composed of striated muscle. (Lewis and Stohr: "Text-book of Histology.")

On the borders of the anal valves are 10 to 14 tubercles or papillæ of highly sensitized tissue. These tumors may be seen by everting the anus (Fig. 1) or through an anoscope (Figs. 2 and 3). The pyramidal teats have a pinkish colored base continuous with the mucous membrane below but a pearl-white tip. They are always present although not easily seen, except when hypertrophied and elongated, at which time they appear as saw teeth projections with their bases directed upward and inward, often associated with inflammation or ulceration of neighboring crypts (cryptitis), and may hang down as long slender ribbons, perhaps an inch long, or may be a rounded ball-like tumor easily mistaken for a skin tag or polyp or an anal wart (Fig. 1).

Digitally they feel like hard nodules about the anus. A digital or specular examination is very painful. These papillæ are tactile organs with a special rectal sense, which if destroyed by a Whitehead hemorrhoidal or other operation permits evacuations to occur without warning. The histological examination of these papillæ is very instructive. Several layers of stratified epithelium cover the

FIG. 1.



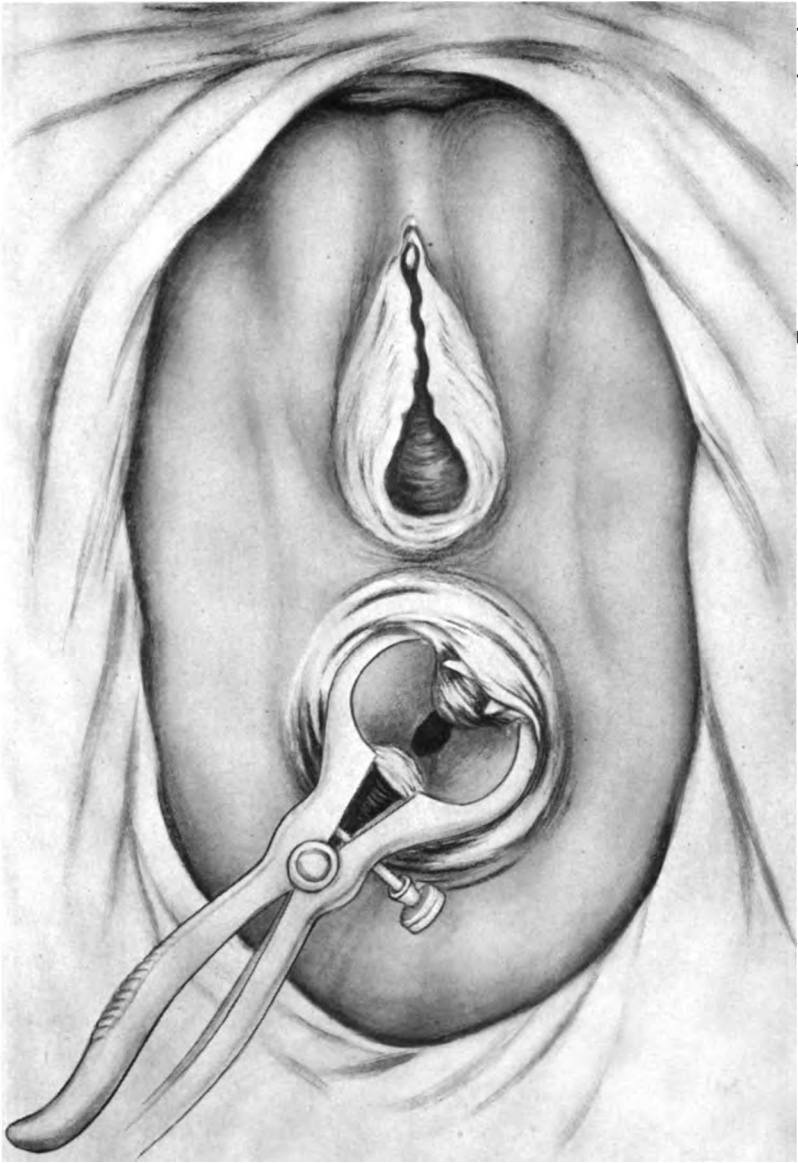
Anus everted showing enlarged papillæ. Also large hemorrhoidal tumor on one side of anal ring.

FIG. 2.



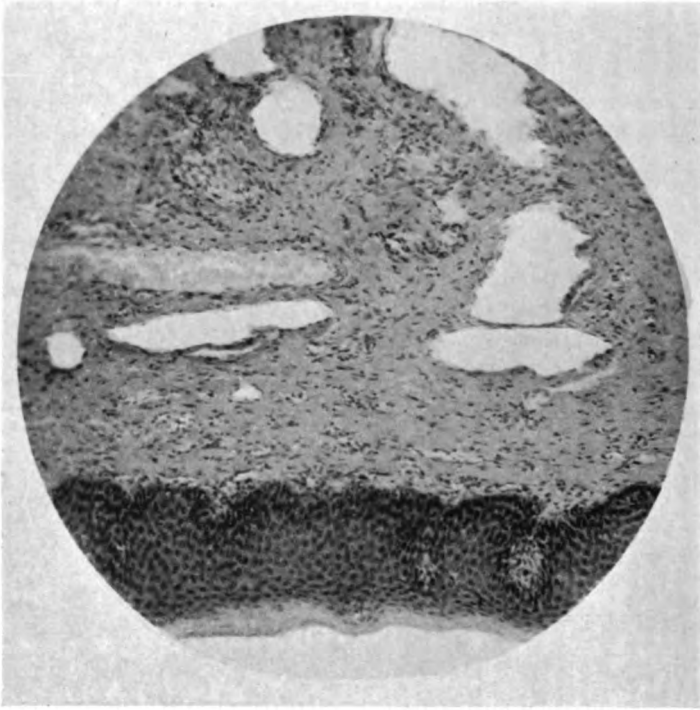
Enlarged papillæ seen through anoscope.

FIG. 3.



Enlarged papillæ seen through operating specula.

FIG. 4.



Microscopical section of hypertrophied anal papillae showing cavernous tissue with stratified epithelium covering.

papilla, the body of which is composed of erectile connective tissue similar to that of the corpora cavernosa of the penis. In this tissue are numerous thin-walled venous spaces with but slender trabeculæ of tissue between them. The veins of this structure have such thick walls as to closely resemble arterioles (Fig. 4). Each papilla has nerve filaments from the pudendal and the inferior hemorrhoidal trunks. Sensory filaments terminate in tactile corpuscles in the superficial or external mucous membrane structures and in lamellar corpuscles in the cavernous tissue.

Etiology.—Why these papillæ become hypertrophied is sometimes very obvious and in other instances quite obscure. It may be caused by traumatism of the anal canal by foreign bodies, hard fæces, repeated replacing of a prolapsing hemorrhoid or polypus, a chronic acrid discharge from cancer, abscess, ulcer or proctitis, digital stretching of prostatic massage, frequent or careless instrumentation, or the rough usage of enema or irrigating tubes.

Symptoms.—Once these papillæ are irritated they set up a chain of neurogenic disturbances which is both painful and otherwise annoying to the sufferer. At each passage of the fæces the cavernous spaces engorge and the papillæ are dragged down through the external sphincter until they protrude externally. There is a feeling of fullness and tickling within the anal canal, a sensation of incompleteness of evacuation, and as the engorgement subsides and the papilla recedes a sensation as though worms were being voided or were crawling on the skin about the anus. There is also a consciousness that the anal sphincter is unduly contracted. This séance recurs with each defecation and it may be several minutes or perhaps an hour until by sphincteric compression and by retraction of the mucosa, the papillæ are depleted and assume their former size and position, when the distressing symptoms subside. As the papillæ become hypertrophied and are continuously enlarged the symptoms are more likely to be constantly present. In some instances the anal sphincter is so spasmodically contracted as to prevent a complete evacuation and the lower rectum remains filled with fæces. An enema given at this time will empty the bowel but its administration is painful and therefore objectionable to the patient. Sometimes lumbar or sacral ache is complained of, also vesical spasms and pains down the legs.

Although the symptoms of papillitis are quite constant this con-

dition is frequently overlooked and treatment instituted for pinworms, hemorrhoids or pruritis, or perhaps the feces are examined for some irritating factor. Such treatment of course fails to relieve, and the patient is then dismissed as a neurotic when his trouble is all local and really very amenable to treatment. If untreated these papillæ may become definite polypoid tumors or the venous congestion be the forerunner of hemorrhoids.

Treatment.—All of this troublesome chain of symptoms will be relieved magically by amputation of the papillæ, which is very satisfactorily accomplished under local anæsthesia. The width of the anæsthetized field may be modified according to the extensiveness of the disease in the case on hand. (My technic of regional anæsthesia I have described elsewhere.)

When but a few individual papillæ are to be removed the infiltration may be confined to the diseased masses. The needle is inserted into the mucous membrane at the base of the tumor and gradually advanced toward the apex, injecting as we proceed. Ten drops of anæsthetic solution to each papillæ is usually enough. When a number of papillæ are enlarged and the sphincter is hypersensitive it is better to anæsthetize the whole anal ring and the sphincter muscle.

Having anæsthetized the papillæ so that it may be manipulated without causing pain, it is drawn out with tissue forceps and cut off well below its base. The wound is not sutured. Local asepsis (careful cleansing after each defecation and a warm sitz-bath each day) is all the after-treatment required. When the patient is discharged he is instructed to continue the ablutions.

Case A., thirty-seven, is forty-five years of age, and the mother of three children. She has always enjoyed good health and has had no bowel trouble. For two and a half years she has had pain, itching and a creeping sensation at the anus, is constipated and feels as though evacuation is incomplete. Anus feels sore. On examination there were found five hypertrophied anal papillæ and one hemorrhoid on the right posterior quadrant of the anus. They were all removed under local anæsthesia. Three weeks later patient reports that she "enjoys the comfort of going to stool and getting results she has not had for years."

COMPARISON OF THE CORTEX CELLS IN CASES OF STARVATION, THIRST, EXCITEMENT, EXTREME PHYSICAL EXERTION, SHOCK AND COLLAPSE

By GEORGE S. FOSTER, M. D.

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Manchester, N. H.

THE study of the cerebral cortex cells, as well as the cells of other soft structures of the body, has been beset by many obstacles and required a great deal of patience. As in previous investigations, along these lines, I have used guinea-pigs for subjects.

These investigations were instituted for the purpose of determining the degree of sensitiveness which the cortex cells showed under varied extraneous influences. The writer has often wondered just how influential the influences herein enumerated would affect bodily economy from a surgical point of view.

Every day we see surgical conditions in patients who are not only affected by the pathological entity amenable to surgical interference, but also are in a state of physical being brought about by other factors. For instance, we see gastric cases which have been more or less upset, not from the pathology in the stomach as such, but rather from the resulting influences or phenomena, such as continued vomiting of a more or less passive nature. Possibly this emesis has been intermittent either regularly or irregularly, or of the type which is regular and frequent, as a daily occurrence *post cenam*. Whatever these signs and symptoms may be, it has often occurred to me to wonder just what influence such factors would have upon the general bearing of the case. All surgeons meet cases which are referred to us in a seemingly very good physical condition. In other words, they are slated as good surgical risks, but now and then one of these cases will be lost without any apparent definite cause. Why did they die? The clinical chart did not indicate anything unusual. They showed a good general average progress, but about the third, fourth, or fifth day something happened. All in surgical work have met these cases, but we are too prone to forget the example they set, and the teachings at hand.

These occasional unfortunate endings cannot be placed at the door of internal secretions or ductless glands in every instance, although, no doubt, very many of these cases are so influenced, yet it seems lack of vital interest not, at least, to try and determine a more definite reason. Then again, there are patients who are extremely hypersensitive and have an exaggerated nervous system. These cases, on the whole, make a fairly good recovery, yet they will run a slightly variable course. Too often, we place the trouble at the door of nerves. Of course, the nervous system plays a most important part, yet we must not be too easily satisfied by this explanation. We should go farther and endeavor to determine the underlying principle if there is one. The patient is first, and we must always study the pre-operative life of the patient as we would a mathematical problem. Is there anything in his life that prevents classification as a good surgical risk? Is there any influential factor inculcated by inherent or acquired qualities which we have not sought out? Are we careful enough in summing up each case pre-operatively? We owe it to our patients to think these matters over seriously.

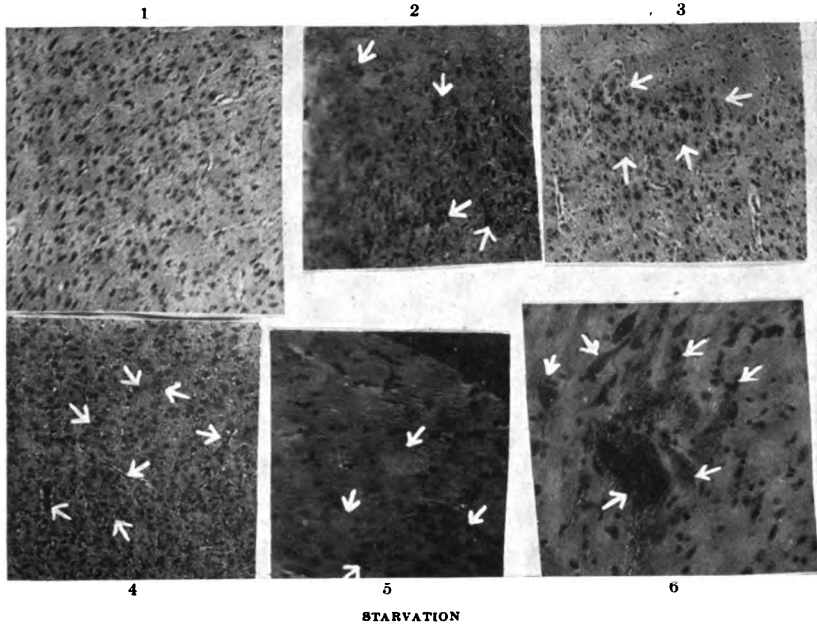
The late Professor Agnew has remarked in his clinics: "Gentlemen, you remember that simple, plain, apparently normal surgical entity upon which I did a hysterectomy last week? Yesterday she died. Why did she die? I do not know. It has been my experience that now and then a purely frank, clean surgical case will step out without any definite reason." Professor Agnew was surely wise in his conclusions, but progress has been rapid and steady since the time of this great Philadelphian, and now it should always be known definitely why a surgical case, apparently a very good risk, did not get well.

Merely for the purpose of self-satisfaction along these lines, I have carried out this study. Although the end has not yet been reached, I feel that some progress has been made, and that I am more fit to save these cases. The conclusions are given, and the facts are placed squarely and fairly before you.

STARVATION

This altered physiological condition, if long continued, would result in true pathology. Starvation has wide areas of cellular influence. Within a certain period the cells attain only a physiological alteration. We are all familiar with the fasting episode of old Doctor

FIG. 1.



Influence on Cerebral Cortex Cells

(Fig No. 1.) Three-day starvation period. Cortex cells unchanged. Pain nerve transmission nil.

(Fig No. 2.) Six-day starvation period. Cortex cells very slightly nephelated and then only discretely. *Note:* No cells fully nephelated and no confluence is seen. Arrows indicate partially affected cells. Pain nerve transmission nil.

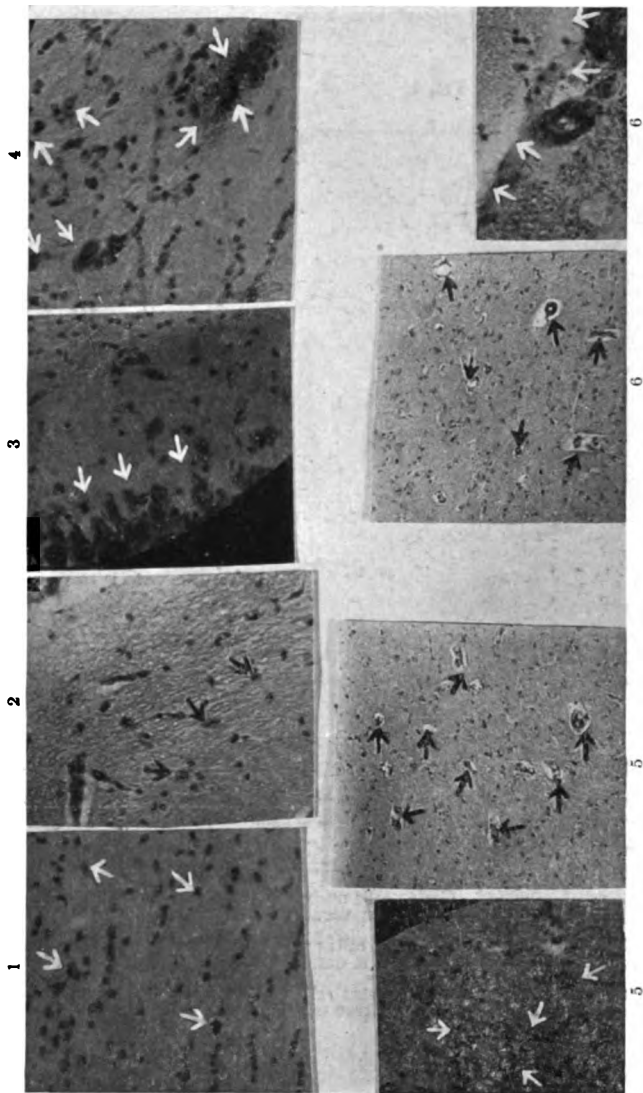
(Fig No. 3.) Eight-day starvation period. Cortex cells discretely nephelated. A few cells are seen here and there fully nephelated. No vacuolation present. Pain nerve transmission practically nil or only fourth degree slightly.

(Fig No. 4.) Ten-day starvation period. Cortex cells nephelated to a marked degree. Confluent nephelation present and some attempt at partial vacuolation. Pain nerve transmission of third degree.

(Fig No. 5.) Twelve-day starvation period. Cortex cells nephelated to complete confluence. Vacuolation marked. Some attempt at complete cellular destruction. Pain nerve transmission of second degree.

(Fig No. 6.) Fourteen-day starvation period. Cortex cells confluent nephelated. Much vacuolation. Cellular destruction in some areas. Pain nerve transmission of first degree.

FIG. 2.

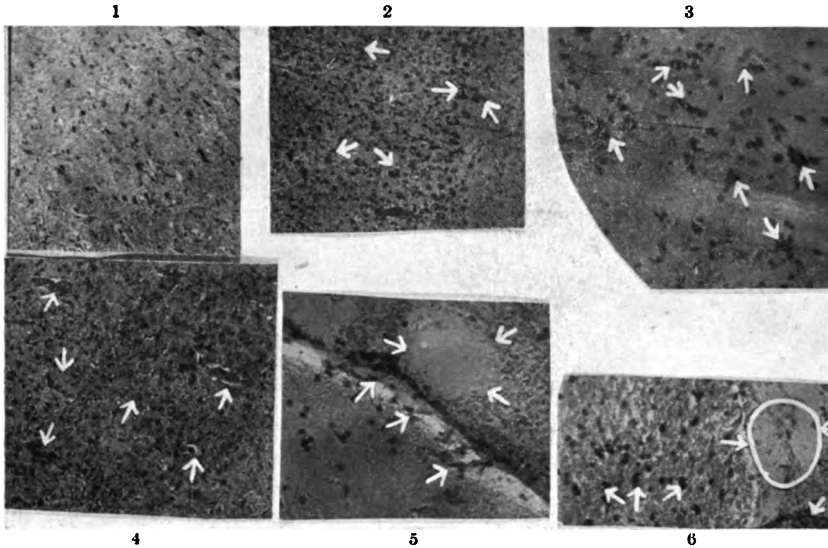


THIRST

Influence of Cerebral Cortex Cells

(Fig No. 1.) Second day. Pain nerve transmission of fourth degree. Note: Nearly all cells normal. Occasional partial nephelation as indicated by arrow heads.
 (Fig No. 2.) Third day. Pain nerve transmission of third degree. Discrete nephelation only. No vacuolation.
 (Fig No. 3.) Fourth day. Pain nerve transmission of second degree. Confluent nephelation and vacuolation.
 (Fig No. 4.) Fifth day. Pain nerve transmission of first degree plus. Marked nephelation. Confluent vacuolation. Not produced in any previous tests.
 (Fig No. 5.) Sixth day. Illustration No. 1. Nephelation (confluent). Illustration No. 2. Vacuolation (confluent).
 (Fig No. 6.) Seventh day. Pain nerve transmission of first degree, double plus. Note: Confluent vacuolation in illustration No. 1, and complete destruction of cells and desiccation in illustration No. 2.

Fig. 3.



EXCITEMENT

Influence on Cerebral Cortex Cells.

(Fig No. 1.) Kept under non-disturbing influences for one week. Pain nerve transmission of fourth degree. Cells normal.

(Fig No. 2.) Kept perfectly quiet one week. Fifteen minutes previous to administration of lethal dose of morphine, an alarm clock was set off in the pen. This was augmented by loud tapping on a tin pan during the last minute. Pain nerve transmission of third degree. Occasional discrete nephelation.

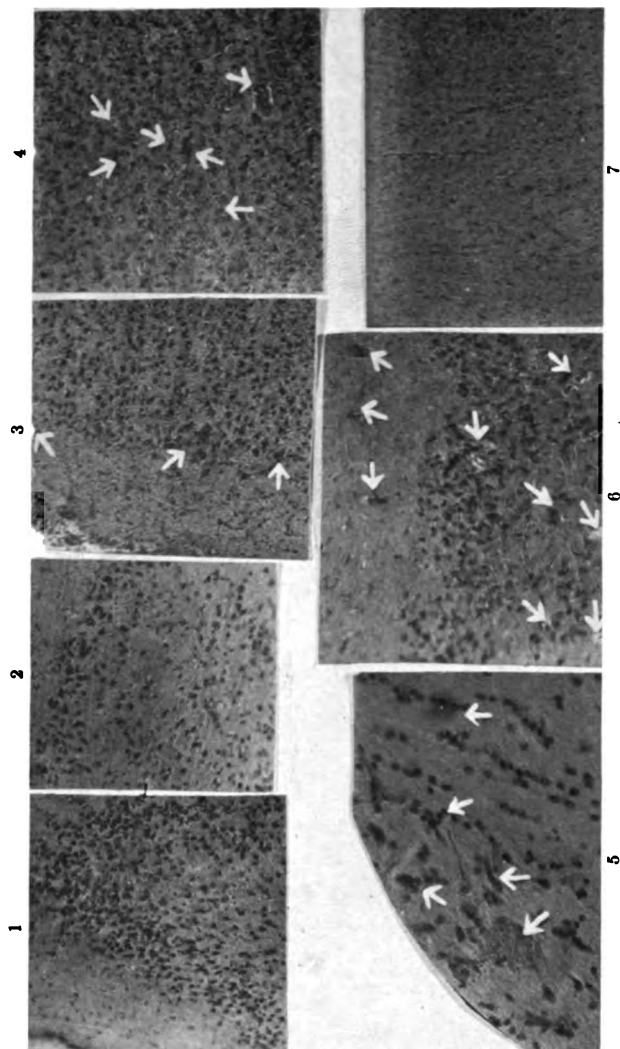
(Fig No. 3.) Excitement stage produced by combination of alarm clock and tapping on a tin pan continuously for ten minutes. Lethal dose of morphine. Pain nerve transmission of second degree. Nephelation to the point of confluent formation without vacuolation.

(Fig No. 4.) Excitement produced by putting pig in a dark, closed box and dragging this box about on a cement surface for fifteen minutes. Occasional pounding upon the sides of box. Nephelation to the point of confluence and a high degree of vacuolation. Pain nerve transmission of the first degree.

(Fig No. 5.) Excitement produced by freeing the pig in a large room and while producing loud noise, chasing him about for twenty minutes. Cerebral cortex cells doubly nephelated and vacuolated. Pain nerve transmission of the first degree double plus. Never before produced in previous experiments.

(Fig No. 6.) Excitement produced as in experiment No. 5, except continued for one hour. Cerebral cortex cells markedly nephelated and vacuolated. Some cells completely destroyed. Pain nerve transmission first degree four plus. Most typical case produced in this set of experiments. Circular area entirely destroyed.

FIG. 4.



EXTREME PHYSICAL EXERTION

Influence on Cerebral Cortex Cells

Six normally healthy pigs on average weight were placed in a cage so constructed as to be kept in continuous motion, a pig was removed and given a lethal dose of morphine. Every fifteen minutes

(Fig. No. 1.) Lethal dose of morphine. Pain nerve transmission nil, after fifteen minutes' vigorous exercise. Normal cells.

(Fig. No. 2.) Lethal dose of morphine. Pain nerve transmission nil, after thirty minutes' vigorous exercise. Normal cells.

(Fig. No. 3.) Lethal dose of morphine after forty minutes of vigorous exercise. Beginning fatigue. Pain nerve transmission of third degree. Here and there a partially or completely nuchelated cell is seen.

(Fig. No. 4.) Lethal dose of morphine after one hour of vigorous exercise. Continued fatigue. Pain nerve transmission of second degree. Cortex cells confluent nuchelated to a marked degree. Compare with illustration No. 3. Pig getting tired out.

(Fig. No. 5.) Lethal dose of morphine after one hour and fifteen minutes of vigorous exercise. Continued fatigue. Pig very tired, fagged out. Pain nerve transmission of first degree. Confluent nuchelation to the point of vacuolation. Compare with Nos. 3 and 4.

(Fig. No. 6.) Lethal dose of morphine after one hour and thirty minutes of vigorous exercise. Marked fatigue. Refused to respond to further effort. Pain nerve transmission of first degree, slightly modified beyond usual limits. Vacuolation slightly greater than in No. 5.

(Fig. No. 7.) Control. Vigorous exercise for one hour and thirty minutes. Complete rest for 24 hours. Lethal dose of morphine. Cells normal. Complete recovery. Compare with No. 6.

Tanner who went one month without food. He lived to a ripe old age, so it cannot be said that this fasting was deleterious. However, we may think of what could have happened if Doctor Tanner had undergone a surgical interference in the middle of this fast. The outcome might have been the same, or it might have been fatal. Fortunately for us, the bodily economy has its strongest fortification in the lymphatic circulation. This is the last system to give way *ante mortem*. In fact, in the majority of cases, it never breaks down altogether.

To illustrate this section I selected six healthful pigs, and put them to the test. The starvation period covered fourteen days.

Pig No. 1 was not allowed food for three days.

Pig No. 2 was starved for six days, and each of the successive pigs, namely, Nos. 3, 4, 5, and 6, were, respectively, not permitted food for eight, ten and fourteen days. At the end of each period, the guinea-pig was given a lethal dose of morphine, and the cortex tissue very carefully removed and preserved. During all of the starvation period, the pigs were given all of the water they desired. No special attention was given to bowel evacuation, although it was noted they all eliminated freely of both urine and stool.

Pig No. 1. Three-day starvation period. Cortex cells unchanged.

Pig No. 2. Six-day starvation period. Cortex cells very slightly nephelated and then only discretely. No cells were fully nephelated and no confluuation was found.

Pig No. 3. Eight-day starvation period. Cortex cells discretely nephelated. A few cells were seen here and there fully nephelated. No vacuolation was present.

Pig No. 4. Ten-day starvation period. Cortex cells nephelated to a marked degree. Confluent nephelation present and some attempt at partial vacuolation.

Pig No. 5. Twelve-day starvation period. Cortex cells nephelated to complete confluuation. Vacuolation marked. Some attempt at complete cellular destruction.

Pig No. 6. Fourteen-day starvation period. Cortex cells confluent nephelated. Much vacuolation. Cellular destruction in some areas.

Therefore, I conclude that starvation produces the same result in cortex cells as does pain nerve transmission, resulting in a varying

degree of shock ; also that a certain amount of pain nerve transmission goes on after a certain period is reached in starvation. This is proved by the fact that these pictures give us for all general purposes the same marks as we find in our studies of pain nerve transmission. The cobweb construction outlined in the article covering this phase in the CLINICS, vol. iii, 28th series, could very well be used here. These are the same cortex cellular changes as are found in pain nerve transmission of the first, second, third and fourth degree.

Guinea-pig No. 1, pain nerve transmission *nil*.

Guinea-pigs Nos. 2, and 3, pain nerve transmission *nil*.

Guinea-pig No. 4, pain nerve transmission of third degree.

Guinea-pig No. 5, pain nerve transmission of second degree.

Guinea-pig No. 6, pain nerve transmission of first degree.

It would seem from these facts that actual pain nerve transmission does occur, so that starvation seems like an inflicted wound, and that the patient is a poor surgical risk in the direct ratio of the limit of starvation or deprivation beyond the third day.

THIRST

It is most interesting to note the influence of a general deprivation of bodily moisture on the cerebral cortex cells. In fact, the results obtained from this phase of the observations herein noted do not differ materially from previously published articles in regard to cerebral cortex cell nephelation as a result of direct pain nerve transmission.

In a previous article, I have cited how nature will supersaturate all cells of the body to prophylactate shock. This applied not only to surgery, but also to medicine in its broadest sense. For instance, in a case of pneumonia, how often the citation of a concomitant pleurisy occurs, and this is followed by the excessive serological phenomena with effusion. The plural cavity becomes filled with an exudate, and this fluid serves the purpose of supersaturating the cells to forestall or block nerve pain transmission and resulting nephelation. As previously stated in another article, I take the stand that cells supersaturated cannot become nephelated as such, and thus by it can be seen how well any tendency toward influence of pain nerve transmission or shock is averted. The same condition is found in cirrhosis of the liver, pericarditis, excessive skull pressure, severe blows upon the abdominal wall, fractures or innumerable other instituted pathological entities. There is an immediate or remote exu-

dation and the cells are so overloaded with moisture that nephelation is avoided or, in other words, the shock of any detrimental degree is not present. Again, we have the well-known sodium chloride phenomena found in pneumonia, as sodium chloride has much influence on the chemical side of cellular affinity. It would be a most interesting thing to make a special study regarding the nephelation of cells following the withdrawal of this material. Later on I hope to carry out an investigation along this line.

The present phase of our subject results in directly opposite conclusions. The withdrawal of fluids from the diet causes an immediate concentration of the sodium chloride in the cells and as a result we find that nephelation is very marked. Because of this concentration of sodium chloride we find that the deeper cells of the body, or rather the cells going to form the internal organs, take up every bit of moisture they can at the expense of the surface soft tissue cells. No doubt, the evaporation factor has much influence upon the surface soft tissue cells as recondensation aids the cells of the internal organs and we know they are the last to become involved.

Ancient medical history (clinically) has taught us that ingestion of fluids is essential to proper bodily economy. In times of long ago, even water was withheld in such fevers as typhoid, and now the pendulum has swung the other way and medical men tell us that they are giving their patients water and other fluids *ad libitum*.

Observations in the use of the Axillary Sup in all major operations has shown us a fluid-filled cell in comparison to the thirst cell of olden times. Then surgical shock occurred frequently. Now we have almost forgotten its presence and go about our work with indifference, so far as this condition is concerned.

Saturate the cells to the point of ballooning and the shock resulting from nephelation is lacking. Drink! Drink! Drink! is the motto of our operating room, and of the patients' room pre- and post-operatively. If shock and nephelation are absent, there is no thirst.

In carrying out this phase of the article, I chose six mature guinea-pigs of good health. Each pig was given a dry diet and this was carried out for one week. Following the first day, one pig was given a lethal dose of morphine daily, so that we have citation on the pigs for each successive day. The cortex cells showed microscopically as follows:

Guinea-pig No. 1, 2nd day, pain nerve transmission of fourth degree.

Guinea-pig No. 2, 3rd day, pain nerve transmission of third degree.

Guinea-pig No. 3, 4th day, pain nerve transmission of second degree.

Guinea-pig No. 4, 5th day, pain nerve transmission of first degree.

Guinea-pig No. 5, 6th day, pain nerve transmission of first degree plus.

Guinea-pig No. 6, 7th day, pain nerve transmission of first degree double plus.

As seen from these citations the absence of fluid ingestion early causes pain nerve transmission, and its resulting nephelation. This degree of alteration is carried far beyond anything I have previously seen, and therefore I have designated the findings on the sixth and seventh days, respectively, as plus and double plus. The cortex cells of these last two days showed on the sixth day confluent vacuolation, something I have previously been unable to produce by any surgical procedure, no matter how long continued. Those of the seventh day showed not only a confluent vacuolation, but also a complete desiccation to the point of peripheral distinction almost like parchment. Just how much, if any, surgical interference these last two pigs could have stood is not much of a question, although undetermined by actual citation. However, it can be clearly seen that thirst early causes pain nerve transmission, resulting in nephelation, bringing on fatigue, shock and collapse.

It is interesting to note that the hair of these pigs became dry and bristly, the cornea cloudy and the sclera wrinkled. It was also noted that no urination occurred after the third day. The internal organs such as the liver, heart, lungs and spleen were soft and friable almost like sand. The kidneys were friable but contained some moisture. The illustrations will clearly show the marked influence upon the cerebral cortex cells.

This very vividly illustrates the need of moisture in cells to withstand any pain nerve transmission with the resulting nephelation.

EXCITEMENT

This element of the nervous phenomena seems paramount at times. As a matter of fact, excitement is but a step in advance of the ordinary nervous status of any individual at all times. The minimum degree of this nervous function is experienced during sleep. Although, even then, it may not remain always at a minimum. Patients may be found asleep, yet physically active to a certain degree, and this physical activity (necessarily) is actuated by a culmination of nervous

impulses. A person in a dream and making movements with the arms or lower extremities illustrates this very well.

The medium degree of excitement is exhibited by all individuals during the wakeful hours of everyday life, since a certain medium degree of excitement is essential for ambition. While this medium degree varies much in individuals, these must be in a certain medium degree of excitement, in order to cause the physiological functions of life.

The maximum degree of excitement is found in some individuals nearly all the time during the wakeful hours. Nervously high-strung, they wear down quickly each day, are generally very active in business matters, and in the majority of instances are fairly successful. However, from a surgical point of view, this class of patients is not a good surgical risk. Generally, it is noticed in our clinic, that such patients require more narcotic pre-operatively, that they take more general anæsthetic than the ordinary case. Possibly, this is more true during the first few moments after the anæsthetic is begun, and that their respirations are more labored but not as deep. Again, post-operatively, it is found that more side issues or complications appear. These patients are very apt to vomit post-anæsthetically and upset the usual negative phase of this aspect of the case. Also, they do not seem to tolerate pain well, but exaggerate the status of the pain. Any slight degree of irritation seems to augment the interpretation of pain. Rest is another essential factor which seems wanting in these cases. For these last two reasons, this type of case demands more narcotic than is good for the general run of cases. Because of this demand for a narcotic the bowels seem less active, and tympanites bothers more or less. Thus it is seen that the patient who is one of the type of maximum degree of excitement brings on little adjuncts which really aggravate an otherwise smooth convalescence.

To prove these facts with animals was a more or less difficult undertaking. However, I desired to in some way simulate the humans, so I selected six good average weight, healthful guinea-pigs. These pigs were selected very carefully and the degree of excitement was controlled by outside influence.

Guinea-pig No. 1—Pain nerve transmission of fourth degree.

Guinea-pig No. 2—Pain nerve transmission of third degree.

Guinea-pig No. 3—Pain nerve transmission of second degree.

Guinea-pig No. 4—Pain nerve transmission of first degree plus.

Guinea-pig No. 5—Pain nerve transmission of first degree double plus.

Guinea-pig No. 6—Pain nerve transmission of first degree four plus.

NOTE: In carrying out these experiments I have assumed, as in all previous experiments along this line, that any nerve transmissional influence of the cortical or cord type, in so far as the resulting effect on the cell itself was concerned, was the same. This phase of the situation was carefully covered in a previous article in the *CLINICS*, vol. iii, 28th series. Here it was shown that any nerve impulse involving cortex or cord had some influence shown by the resulting individual cell reaction. All these pigs were killed by a lethal dose of morphine.

Guinea-pig No. 1 was kept under very non-disturbing influences for a week previous. As will be seen by the accompanying illustrations, no influence was shown by reaction in the cortex cells, so that the cells were perfectly normal.

Guinea-pig No. 2 had been kept perfectly quiet for one week. Fifteen minutes before the morphine was given, an alarm clock was set off in his pen, and just previous to the morphine, there was loud tapping on a tin pan for one minute. This pig became quite excited, and apparently had great fear. In these experiments we include the influence of apparent fear shown by a desire to get under cover as a part of the excitement. To differentiate these two factors would seem quite impossible, both in the method used to produce them and to the degree of each. The accompanying illustrations will show cortex cells of the third degree nerve transmission where nephelation was produced only to a slight degree and then only discretely.

Guinea-pig No. 3 passed through a stage of excitement lasting ten minutes. This excitement was produced by an alarm clock and pounding upon the pan with a stick of wood. All external appearances indicated that the pig was greatly excited. He desired to seek a secluded part of the hutch and at times would run about bewildered. The accompanying illustration will show the cerebral cortex cells of pain nerve transmission of the second degree, or nephelation to the point of confluent formation but without vacuolation.

For guinea-pig No. 4, the exciting stage was produced by putting the pig in a dark, closed box and dragging this box about on a hard cement surface for fifteen minutes. The box was roughly handled, but not in a way to traumatically injure the pig. Occasionally the

box was pounded with a stick. The pig became very much excited and upset. The accompanying illustration will show the cerebral cortex cells markedly nephelated to the first degree of pain nerve transmission. Nephelation was marked, confluent, and a high degree of vacuolation was present.

For guinea-pig No. 5, the excitement was produced by freeing the pig in a large room, and while producing loud noise, chasing him about for twenty minutes.

The question might arise as to the production of fatigue in this instance. It might be stated that after the extreme degree of excitement had been produced, the pig was in no wise tired. He appeared vigorous and strong, yet was very excited. Here, again, as in the case of pig No. 2, it would be somewhat difficult to differentiate the influence of excitement from that of fatigue. However, credit is given to the former for any cortex cell injury because of the vigorous condition and good health of the pig, as there seemed to be no better way to differentiate. The accompanying illustration will show the cerebral cortex cells of this pig just doubly nephelated and vacuolated as compared to pig No. 4. I have thus standardized it as first degree double plus, a degree which I have never been able to produce in previous experiments.

Guinea pig No. 6 was killed with a lethal dose of morphine. The same experiment was carried out as in the previous case, except it was continued for one hour. Fatigue did not seem to enter into the circumstances as the pig was good and strong at the end of the experiment. The alarm clock, pounding, continued actuation, and other exciting methods were used. The illustration will show the cerebral cortex cells very markedly nephelated and vacuolated. Some even completely destroyed and greatly influenced. I have standardized this as pain nerve transmission of the first degree four plus, the most typical case yet produced.

RÉSUMÉ

As is shown by the illustrations, excitement has much influence upon the reactionary stability of the guinea-pig. When the guinea-pig was kept under very quiet surroundings for some days and death then produced by a lethal dose of morphine, pain nerve transmission was of the fourth degree. The cortex cells were found normal in

every respect. If the illustrations are carefully studied, it will be seen that if the degree of excitement is increased, so will the pain nerve transmission resulting from any procedure also be augmented. The purpose was to excite and not to produce fear or fright. To differentiate these by cortex cells influence is rather a delicate procedure, yet the excitement was the thing sought and the endeavor made to produce it. The point is that the guinea-pig that was not excited at all, as in case of No. 1, could much better stand any operative procedure than could any of the others where the degree of pain nerve transmission had been present to the point of single or multiple degrees.

The argument shows that any patient who goes to the operating room in any degree of excitement does so carrying a weight which is unnecessary and should be averted. Anything which produces pain nerve transmission to any degree will burden the patient with a longer convalescence, and should be avoided. The one exception exists in emergency cases which prove the rule by the form of convalescence.

EXTREME PHYSICAL EXERTION

It is often necessary to perform operations of a strictly emergency nature, upon patients who have just previously or for a greater or less length of time previously undergone more than the ordinary amount of physical exertion, as, for example, an athlete who has for days been training for some special meet. This young man going into the fray fit must exert himself to the limit if he would win. He enters a contest for a hundred-yard dash or a mile run. Every muscle is brought into play and extreme exertion excited against his rivals. Near the finish he falls and acquires a Colles' fracture, which immediately disables him, and necessitates surgical skill. He is given a general anæsthetic and the fracture reduced. Ordinarily the convalescence, in so far as ambulation is concerned, is speedy. However, in this special instance, this young man awakens from his slumber to find that his strength is greatly reduced. He is prostrated, and it takes some days ere he feels able to be up and about. Was it the fracture that produced this condition or was it the physical exertion to an extreme degree which he had previously made? As the ordinary case of Colles' fracture will be up and about immediately, we must assume that the extreme degree of physical exertion had some-

thing to do with it. At least, by exclusion, this factor should be considered as one to be reckoned with.

The writer clearly recalls a young man who made the All-American centre on the football team. This man was a marvel of physical make-up, trained to the minute. He went through an extremely hard-fought contest and subsequently developed a fulminating type of appendicitis. Immediately he was operated upon and the case was one where the ordinary individual would recover *per se*. This young man post-operatively was markedly prostrated and very quickly succumbed to the disease. Whether he would have recovered had he not gone through this extreme degree of physical exertion will never be known. However, we can assume from the following conclusions of the experiments on guinea-pigs, that he certainly would have made a better fight.

Just what influence extreme physical exertion has on producing pain nerve transmission will be clearly shown in the following illustrations taken from specimens produced in guinea-pigs' cortex cells.

In carrying out the experiments on this phase of the subject, six healthy, normal, average weight guinea-pigs were selected with great care in order to see that each was of equal weight and age. Previous to the experiments they were kept under normal surroundings and fed well. They were taken in order and consecutively given fifteen minutes of additional exercise. It was rather a difficult thing to do this and not produce fright or excitement. After considerable experimenting, a plan was devised whereby the pigs were kept on the move by motion of the cage itself and angular planes were made so that the pig must climb as well as walk or run on a level. Every fifteen minutes a pig was removed and given a lethal dose of morphine. The findings in the cerebral cortex cells are noted in the table showing the degree of pain nerve transmission. These were killed by a lethal dose of morphine. Guinea-pig No. 1 after fifteen minutes' vigorous exercise. Cortex cells show pain nerve transmission of *nil* degree. Guinea-pig No. 2, thirty minutes' vigorous exercise. Cortex cells show pain nerve transmission of *nil* degree, perfectly normal, no fatigue.

Up to this point the pigs had rather seemed to enjoy the work, but now it was noticed that they were showing signs of desiring to seek rest and not to work longer. First end (normal cell) control. Guinea-pig No. 3, forty-five minutes' vigorous exercise. Cortex cells

show pain nerve transmission of third degree. Illustration will show the cortex cells not entirely involved, but here and there a partially or completely nephelated cell is found. Guinea pig No. 4, one hour vigorous exercise. Cortex cells show pain nerve transmission of second degree. Illustration will show the cortex cells confluent nephelated to a marked degree. The extent of change of cortex cells, as shown by comparing this illustration with that of pig No. 3, is very striking. The amount of pain nerve transmission shown by the nephelation which had taken place in the last fifteen minutes of the hour is very emphatic. This pig was getting tired, and the effect is very apparent. Guinea-pig No. 5, one hour and fifteen minutes' vigorous exercise. Illustration shows the cortex cells confluent nephelated to the point of vacuolation. Here again the degree of cortex cell alteration is very marked compared to the previous illustration. This pig was nearly fagged out and apparently about "all in." He sought and needed rest. Guinea-pig No. 6, one hour and thirty minutes' vigorous exercise. Cortex cells show pain nerve transmission of first degree. Illustration shows the cortex cells confluent nephelated to the point of vacuolation. This illustration, as compared to the previous one, does not show any marked differences except possibly the vacuolation is more numerous. This pig was very tired and would hardly respond with further effort.

RÉSUMÉ

Several points are clearly brought out by this experiment. First: It is clearly shown that the average individual can endure certain vigorous exercise without any apparent influences upon the cerebral cortex cells. Second: As soon as fatigue begins to show itself the cerebral cortex cells show pain nerve transmission in the form of nephelation. Third: The ratio of degree of pain nerve transmission compared to the length of time of continued vigorous exercise holds no staple scale. In other words, the degree of pain nerve transmission, as shown by nephelation, is not increased on a ratio with the length of time or vigor put with the exercises. Fourth: Up to a certain point no cortex involvement was present. A continued fifteen-minute period caused a marked pain nerve transmission and the third degree was reached, the fourth degree being skipped. In other words, when the exercise is sufficient to bring on fatigue, the cortex cells change

quickly and profoundly. Therefore, it can be seen that up to a certain point the cortex cells are immune. Beyond this point they become quickly involved and are in compound ratio to the length of time or amount of exercise. As a fifth point, it is suggested from these experiments, that is, so far as glycogen can be supplied in sufficient amounts to satisfy the muscular effort, just so far are the cortex cells protected, and the pain nerve transmission shown by nephelation is *nil*. When this glycogenic influence ceases or is lowered immediately fatigue shows itself and the cortex cells have no protector, and control influence is lost. Sixth point is that given a patient in the condition of guinea-pigs Nos. 4, 5 and 6; the question arises, How would they withstand any degree of surgical interference without previously resting? If they enter as an immediate and urgent emergency, demanding surgical interference, would they pull through following this degree of fatigue? On the other hand, being admitted as an immediate, urgent emergency surgical entity, would they not withstand the procedure better? It would seem that the former instance, in part at least, accounts for the now and then unaccountable surgical death as spoken of by Agnew years ago. At least this is a logical thought upon which to base more conservative action in these fulminating or other cases. It should lead one to be very careful in making inquiry as to the occupation and specific amount of work and hours spent for a week previous.

A seventh pig was run along with the six tabulated in this experiment, but no previous note made of the fact as he was not used for the specific table. However, when pig No. 6 was killed this pig No. 7 was put at rest, and after a twenty-four-hour period was killed with a lethal dose of morphine. Illustration will show that the cerebral cortex cells of this pig were perfectly normal. Not even the extra cellular adventitia shows any oedema or nephelation. In just what period of time these cells recovered is not known. However, it does show that a twenty-four-hour period of rest will work wonders. At the time this pig was killed he had fully recovered from his fatigue and was voluntarily exercising and had enjoyed a good meal. This would bear out the contention that rest previous to any surgical intervention is profitable to the patient. Pig No. 7 would have apparently stood any average surgical interference well after the rest period. It also shows how quickly pain nerve transmission shown by nephelation

is overcome when resulting from extreme physical exertion. No doubt the glycogen protectorate has much to do with this status. It would be worth while to carry out chemical investigation along these lines. Possibly glycogen had nothing to do with it, but apparently its absence or lack in sufficient amount was an influencing factor in the fatigue produced after the thirty-minute period, and this absence was potent in so far as the cortex cells show. This potency was multiple in its ratio beyond this point of time.

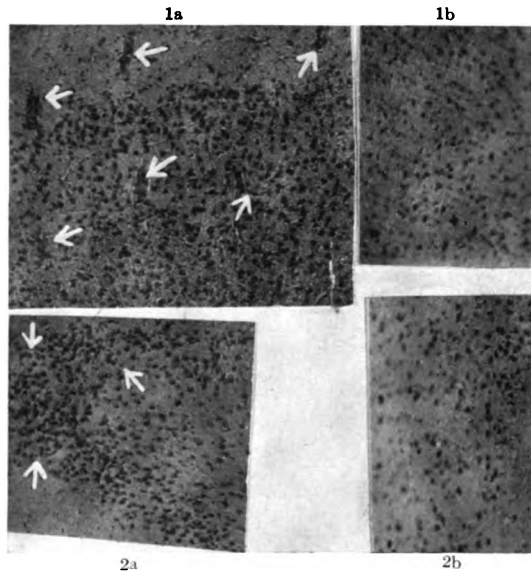
SHOCK AND COLLAPSE

To define these two important factors in surgery is a difficult problem. In themselves they occupy much space, and unlimited papers could be written, and have been done. This paper merely treats with the conditions in so far as their effect shows in the cerebral cortex cells.

Therefore, for a working basis, shock, which is a condition brought about through the nervous system actuating one or more of its divisions, has been chosen. On the other hand, collapse as a condition brought about solely through the actual loss of blood from the circulating channels is the basis. From this differentiation, the effects of each upon the cerebral cortex cells can be taken up. It can readily be seen that a local or semi-general temporary ischæmia produced by the vasomotor nervous system influence might be the result. This could be considered an internal bleeding without any of the blood fluid elements escaping to any degree from the circulating channels. Death from chloroform, it seems to me, is an illustration of definite shock, as the subject dies because in reality he is bled to death in his own blood channels. Hare found from his substantial investigations as a member of the Chloroform Commission that the vasomotor influence caused a marked dilatation of the central circulation channels below the diaphragm and the subject was internally and intra-channelly bled to death. In other words, there is a definite and more or less permanent ischæmia of all of the higher nervous tissues, and death results from the occasional paralysis of the respiratory centre.

Working on this basis I endeavored, after considerable thought, to work out two experiments to show the effect of shock and two experiments to show the effect of collapse upon the cerebral cortex cells.

FIG. 5.



SHOCK AND COLLAPSE
Influence on Cerebral Cortex Cells

SHOCK

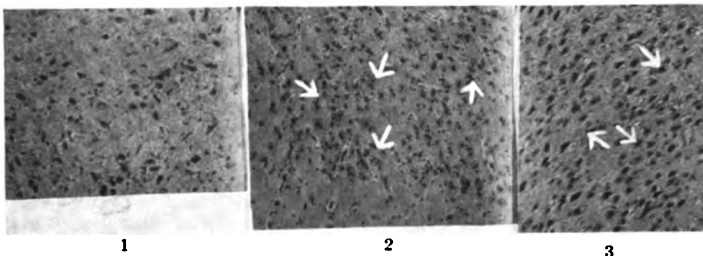
(Fig No. 1a.) Fed ten drops of tincture of ergot, three times a day for one week. Pain nerve transmission of first degree. Confluent nephelation and vacuolation.

(Fig No. 1b.) Same treatment as Fig No. 1a. Forty-eight-hour interval after cessation of ergot administration. Pain nerve transmission nil. Cortex cells normal. (Control citation.) Compare with No. 1a.

(Fig No. 2a.) Exercise, under quiet conditions, on a greased glass surface for one hour. Sympathetic nervous system brought into play. Pain nerve transmission of second degree. Confluent nephelation.

(Fig No. 2b.) Same treatment as Fig No. 2a. Twenty-four-hour period of rest at conclusion of experiment. Lethal dose of morphine. Cerebral cortex cells normal. Fully recovered. (Control citation.) Compare with No. 2a.

FIG. 6.



COLLAPSE

(Fig No. 1.) 60 c.c. of blood withdrawn from the left ventricle. Lethal dose of morphine after one hour intermission. Pain nerve transmission of fourth degree. Nearly all cells normal. Minimum test.

(Fig No. 2.) Same as No. 1, except 120 c.c. of blood withdrawn. Pig became very weak and could not stand. Pain nerve transmission of the third degree. Discrete nephelation (maximum test).

(Fig No. 3.) Both carotids ligated under profound local analgesia. Intermission of 15 minutes. Lethal dose of morphine. Pain nerve transmission of the third degree. Discrete nephelation.

This part of the experimental work was most difficult and had to be carried on with a great degree of dexterity.

To produce shock and to confine it to the limitations of our interpretation, two methods were used. In so doing, in my opinion, two divisions of the nervous system have the most to do with producing this condition, namely, the vasomotor nervous system and the sympathetic nervous system, and a way to show each was found. To produce the former, a guinea-pig was fed on a definite amount of ergot for a definite period. To produce the latter, a pig was compelled to move about upon a greased glass surface. The former brought into action the vasomotor constrictors which to me seemed the most predominating phase causing shock through the vasomotor system. The latter fully exercised the sympathetic nervous system, attempt being made to imitate a person walking upon an icy or slippery surface.

SHOCK

Guinea-pig No. 1 was fed ten drops of tincture of ergot three times a day for one week. Illustration shows cerebral cortex cells in pain nerve transmission of the first degree. This experiment clearly illustrates the influence of shock produced through the vasomotor nervous system on the cerebral cortex cells. Just such a condition of the cerebral cortex cells would be produced from any like constrictor influence. What a poor condition such a person would be in to undergo any surgical procedure! To prove this contention a control pig was given the same treatment but not killed until forty-eight hours after the withdrawal of the ergot. He was then given a lethal dose of morphine. The cerebral cortex cells had fully recovered and were normal. This merely supports the pre-operative rest theory regarding patients admitted in shock. Of course, emergency conditions arise where such a rest cannot be given and the chance against odds must be taken.

Guinea-pig No. 2 was made to exercise, under quiet conditions, on a greased glass surface for one hour. Illustration shows cerebral cortex cells in pain nerve transmission of the second degree. That the overtaxing of the sympathetic nervous system will produce pain nerve transmission resulting in cerebral cortex cell nephelation is clearly shown in these sections. This is merely another way of producing shock. The cortex cells were not as freely involved as in the previous

experiment, yet the degree was but one point less. A control pig was also used in this experiment. He was allowed to rest for twenty-four hours, when a lethal dose of morphine was given. The cerebral cortex cells had fully recovered and were normal. This experiment brings forward the point of determining, if possible, the special system of the nerve anatomy which is involved. The sympathetic system evidently recovers much more readily than do some of the other nerve tracts, so that a shorter recuperating period is indicated. If we can determine definitely the system of nerve tracts involved we can the better and more successfully treat the case.

COLLAPSE

To produce this condition two methods were selected. As previously stated, I believe that collapse is brought about through the actual loss of blood from the circulatory channels. The first pig was bled freely to illustrate the results. The second pig had the carotids ligated which seemed the best method to cause the circulatory loss. Of course, in reality there was actually no loss of blood, but rather a blocking or prevention which seemed the only way to illustrate the desired condition.

Guinea-pig No. 1.—60 c.c. of blood was withdrawn from the left ventricle. Following this the pig was given a lethal dose of morphine. The cerebral cortex cells showed pain nerve transmission of the fourth degree. Thus is illustrated the direct influence of a minimum degree of collapse upon the cortex cells.

Guinea-pig No. 2.—120 c.c. of blood was withdrawn from the left ventricle. Following this the pig was given a lethal dose of morphine. Following the withdrawal of the blood, the pig became very weak and could not stand. This change, which I term collapse, was immediate and very marked. The cerebral cortex cells showed pain nerve transmission of the third degree, which illustrated the direct influence of a maximum degree of collapse upon the cortex cells.

Guinea-pig No. 3.—Both carotids were ligated after careful dissection under profound local analgesia. The pig was left for a fifteen-minute interval, and then given a lethal dose of morphine. During the rest interval, the pig showed no signs of extreme distress, yet he immediately became weak and unsteady. The cerebral cortex cells showed pain nerve transmission of the third degree, which clearly

illustrated the influence of this phase of collapse upon the cerebral cortex cells.

From these four experiments, actuated to produce shock and collapse, it is very definitely shown that to produce pain nerve transmission, it is not necessary to cut or crush; in other words, it is all a nerve phenomena. In fact, anything which might interfere with the physiological economy of the body would have a marked tendency to produce pain nerve transmission and show its effect upon the cerebral cortex cells. So that we are dealing with a pathological entity and the individual resistance is at once lowered. When such a lowering is produced, we find the patient is not a good surgical risk, since he cannot stand any operation as well. The untimely deaths of cases in systematic surgery represent these patients and this is the cause, with one condition excepted, namely, status lymphaticus.

No patient should undergo an operation who has not first gone through the systematic investigation and had the physiological standing rated. The physiological rating should be as carefully gone into as the pathological. We should also take into account just how much harm any pathological lesion has produced. The conclusions drawn should sum up the case as to whether or not it is operable, at that time, unless it is an urgent emergency, or whether it would not be more conservative to wait until a proper stage had been reached. There is always a right and wrong time to operate. If possible, we should select the proper time, and thus give the patient every benefit to enhance the chances for recovery and also thereby shorten the convalescence and make it more comfortable. These are things which are due the patient in every instance.

These few little experiments have been carried out with the idea of showing what may happen under varying influences and what degree of alteration the patient needs. Rest, amount of fluid content of the body, degree of nervous excitement, the amount of muscular effort, and the conditions of shock and collapse have been prudently, conscientiously and diligently considered and weighed.

The findings are herein given as carefully deducted. Each experiment is fully illustrated and deductions drawn. It is for all surgeons in a sense to qualify their individual cases that the risk from operative interference may be lowered to the minimum. Once this is accom-

plished, the full duty is rendered, but anything short of this causes hazardous risks or complete failure.

CONCLUSIONS

1. Proper nourishment and a well-governed digestive system are essential to good results from surgery.

2. Maximum fluid content of the body cells is imperative in order to avoid surgical errors.

3. All patients, when possible, should be given a rest space previous to operation.

4. Extreme physical exertion renders one less fit to undergo any surgical interference. Time for recovery from the results of this exertion should be given whenever possible.

5. Shock is a condition of nervous phenomena which should always be avoided whenever possible.

6. Collapse is a danger signal for any operative interference. Full time should be given for recovery from this condition and the circulatory channels either physiologically or artificially refilled.

7. These conditions have but a transitory influence if the means of actuation in each instance is withdrawn.

8. Every case is a mathematical problem in itself, and should be carefully worked out along these lines.

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